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THE NATURE OF THE REDUCING SUBSTANCE IN THE URINE OF INFANTS WITH NUTRITIONAL DISORDERS *

OSCAR M. SCHLOSS, M.D.

NEW YORK

The occurrence of a reducing substance in the urine of infants affected with gastro-enteric or nutritional disorders has long been recognized. Grosz [1] examined the urine of fifty breast fed infants and the urine of ten reduced Trommer's and Nylander's solutions. In no case did the urine ferment with yeast. Most of the positive results were from infants ill with gastro-enteric disorders. Grosz observed that melituria diminished when tea was substituted for breast milk, and again increased when the diet of breast milk was resumed. He thought that the reducing action of the urine was due to lactose or to a split product of lactose.

Orban [2] concluded that the reducing substance which commonly occurs in the urine of infants with gastro-enteric disturbances is lactose. Tests of the feces in such cases showed the absence of lactase, the ferment which splits lactose into glucose and galatose. It is well known that unaltered lactose cannot be utilized by the organism. If lactose enters the blood stream, it is eliminated in the urine. Due to the supposed absence of the specific lactose splitting enzyme (lactase), Orban considered that unchanged lactose was absorbed with resulting lactosuria.

Langstein and Steinitz [3] examined the urine of thirty-five infants affected with gastro-enteric disorders. They concluded that the reducing action of the urine in such cases is always due, in part at least, to lactose. In a few cases they felt that they demonstrated the presence of galactose in addition to lactose. Contrary to Orban, they found that lactase was present in the intestinal contents of infants with

* Received for publication, Nov. 17, 1920.

* From the Pediatric Service of the New York Nursery and Child's Hospital and the Department of Pediatrics, Cornell University Medical College.

1. Grosz, J.: Jahrb. f. Kinderh. **34**:83, 1892.
2. Orban: Prag. med. Wchnschr. **24**:427, 441, 454, 1899.
3. Langstein, L., and Steinitz, F.: Beitr. z. Chem. Phys. u. Path. **7**:575, 1906.

glycosuria. They, therefore, concluded that the presence of lactose in the urine was not due to absence of lactase but was dependent on some intestinal injury which permitted the absorption of unchanged lactose.

During the past seven years I have examined the urines of infants affected with severe gastro-enteric or nutritional disorders with the object of determining the nature of the reducing agent when such was present. In the beginning, the urines were examined by practically every method which might elucidate the problem. As the work progressed, it became evident that many of the procedures were useless. It was found that an accurate determination of the nature of the reducing substance offered great difficulties owing to the small amount present in the urine and also in many cases to the diminished secretion of urine which is common in the gastro-enteric disorders of infants. Many of the available tests for the identification of sugars in dilute solution are inexact and uncertain when applied to urine containing less than 0.2 per cent. of sugar. This is generally true, but applies particularly to the identification of osazones and to polariscopic examination when only a small percentage of sugar is present. The fermentation test is inexact when the sole criterion is the formation of visible gas. Carbon dioxid is soluble to a degree in urine. The test, however, is of distinct value when used to determine whether the reducing substance is partly or completely destroyed by yeast with the formation of alcohol. In this manner, the test is useful even though the amount of sugar is too small to give rise to visible gas.

Before considering the tests to determine the nature of the reducing substance in the urine of infants with gastro-enteric disorders, it is necessary to consider briefly the question of urine sugar in general. For some time it has been considered probable that all normal urines contain reducing sugar. Recently Benedict and his collaborators [4] have shown that sugar is always present in the urine of normal adults and by means of fermentation with yeast can be divided into two fractions, one fermentable and one nonfermentable. They also found that the amount of urine sugar is dependent largely on the sugar intake. We have found that the same condition pertains to the urine of infants.[5] Since sugar is always present in normal urine, an estimate of its significance must be made on a quantitative basis. The presence or absence of reduction of copper test solutions is not sufficient, as such tests are purely qualitative and may be misleading. If the secretion of urine is less than normal, it can contain sufficient sugar to reduce a copper

4. Benedict, S. R.; Osterberg, E., and Chenowith, I.: J. Biol. Chem. **34**: 217, 1918.

5. Unpublished results of Dr. E. Harrington and myself. Also Greenthal, R. M.: Am. J. Dis. Child. **20**:556 (Dec.) 1920.

test solution and yet the total sugar eliminated may be abnormally small. Reversely, if there is diuresis, the total sugar elimination may be increased in the presence of a negative qualitative test.

For this reason, as pointed out by Benedict,[4] the use of the term glycosuria in its generally accepted meaning is no longer accurate. It has been used to designate the reduction of a copper test solution with the idea that sugar was an abnormal constituent of urine and was detected in this manner Owing to the inaccuracy of the term glycosuria, Benedict has proposed the term glycuresis to designate an increased sugar elimination.

TABLE 1.—DETERMINATION OF THE REDUCING SUBSTANCE BY THE QUANTITATIVE METHOD OF BENEDICT

Name	Age, Mos.	Diet*	Reducing Substance in Urine, Calculated As Glucose, per Cent.	Fermentation With Yeast for 24 Hours		
				Gas Production	Reduction of Benedict's Qualitative Reagent after Fermentation	Nonfermentable Reducing Substance, Calculated as Glucose, per Cent.
A. T.	3½	P. M.	1.1	++	+	Too small to determine
J. A.	4	B. W.	0.95	0	—	Too small to determine
J. Q.	5	P. M.	1.8	++	+	Too small to determine
W. Z.	5	W.	0.6	0	—	Too small to determine
A. B.	6	P. M.	1.63	++	+	0.68
B. Y.	6½	P. M.	2.1	+++	+	0.72
T. J.	7	P. M.	0.63	0	—	Too small to determine
C. M.	8	P. M.	0 22	0	—	Too small to determine
N. C.	8	B. W.	1.16	++	+	Too small to determine
W. T.	8½	P. M.	0.19	0	—	Too small to determine

* The diets given were the ones given during the period of urine collection. P. M. = protein milk (Finkelstein's formula); B. W. = 2 per cent. barley water; W = water.

Eighty-two patients were observed, from whom 196 specimens of urine were examined. All of the patients were affected with severe nutritional disturbances characterized by diarrhea, refusal of food, vomiting or loss of weight. The greater number of patients showed the presence of toxic symptoms. The urine of sixty-three infants reduced Benedict's qualitative reagent. The amount of the reducing substance was determined in sixty-five specimens of urine from thirty-eight infants by Benedict's quantitative reagent, Bertrand's method or the micro-method of Benedict and Osterberg.[6] The amount of reduc-

6. Benedict, S. R., and Osterberg, E.: J. Biol. Chem. **34**:195, 1918.

ing substance calculated as glucose varied from 0.12 to 2.1 per cent. Some of the results are shown in Tables 1 and 2.[7]

The glycosuria in these cases is of relatively short duration, so that repeated examinations of the reducing substances in the urine of the same patient is often impossible. As a rule, glycosuria occurs only during the period of acute illness, and the patient either succumbs or improves within a few days. With improvement, the glycosuria diminishes markedly and subsides. The duration of glycosuria varies greatly, but in most cases covers a period of from one to four days.

TABLE 2.—DETERMINATION OF THE REDUCING SUBSTANCE BY THE MICRO-METHOD OF BENEDICT AND OSTERBERG

Name	Age, Mos.	Diet*	Reducing Substance in Urine, Calculated As Glucose, per Cent.	Fermentation With Yeast for 24 Hours		
				Gas Production	Reduction of Benedict's Qualitative Reagent after Fermentation	Nonfermentable Reducing Substance, Calculated as Glucose, per Cent.
S. C.	3	P. M.	0.582	0	+	0.184
M. D.	3	P. M.	0.163	0	—	0.063
D. W.	4	P. M.	0.5.	0	+	0.192
N. T.	4½	B. W.	0.386	0	—	0.097
A. T.	4½	P. M.	0.479	0	—	0.106
T. J.	5	W.	1,267	++	+	0.410
B. W.	5	P. M.	0.534	0	+	0.146
A. Z.	5	P. M.	0.293	0	—	0.072
D. W.	6½	P. M.	0.736	+	+	0.214
P. J.	7	P. M.	0.496	0	—	0.076
B. D.	7	P. M.	0.638	0	+	0.180
W. M.	7	B. W.	1,347	++	+	0.430
A. E.	8	B. W.	0.920	+	+	0.215
F. J.	8½	W.	0.464	0	—	0.093

* The diets given were the ones given during the period of urine collection. P. M. = protein milk (Finkelstein's formula); B. W. = 2 per cent. barley water; W = water.

The short duration of the sugar elimination in this variety of case is in marked contrast to the continuous glycosuria associated with the regular ingestion of large amounts of sugar. A consideration of the latter type will be the subject of a separate communication.

As shown in the tables, fermentation by yeast always caused a marked diminution or entire disappearance of the reducing substance. In only nine specimens, however, was there visible gas production. These were the ones which contained the largest amount of reducing substance. In order to estimate correctly the amount of the reducing substance destroyed by yeast, it is necessary to consider the delicacy of the tests used. The method of Benedict and Osterberg is sufficiently delicate to show that the reducing power is practically never completely destroyed by yeast. Copper reduction tests are much less delicate and will be negative in the presence of amounts of sugar easily demonstrable

7. These results are from infants showing the largest amounts of urine sugar.

by the method of Benedict and Osterberg. The relation is largely quantitative. As a rule, tests with Benedict's qualitative copper solution are positive when the amount of sugar (estimated as glucose) is approximately 0.1 per cent. This variation in the results obtained with the two methods is apparent by comparing Tables 1 and 2.

These results show definitely that the greater amount of the reducing substance is fermentable and, therefore, they do not agree with the observations of Grosz, Langstein and Steinitz and others who found that the reducing substance was not fermented by yeast. The fact that in my cases the reducing substance was largely destroyed by yeast, indicates definitely that it was not lactose. Whether the fraction of the reducing substance not fermented by yeast was lactose is impossible to say. I am aware of no facts which bear directly on this question. This substance is usually present in amounts too small to determine its nature by the ordinary tests. Moreover, it always occurs in normal urine. It is of interest that in a number of cases the urine after fermentation still reduced Benedict's qualitative reagent (Tables 1 and 2). It is possible that part of this reducing action may have been due to the presence of a small amount of lactose in addition to the usual nonfermentable sugar of urine. Definite tests bearing on this question were not made.

Additional evidence that the reducing substance is not lactose was obtained by preparing osazones from ten specimens of urine. These specimens all contained 1 per cent. or more of reducing substance calculated as glucose so that sufficient osazones were obtained for purification. Their solubility, appearance and melting point indicated that the sugar was not lactose but was probably glucose (Table 3).

TABLE 3.—Appearance, Solubility and Melting Point of Osazones

Name	Percentage of Reducing Substance Calculated as Glucose	Appearance of Osazone	Melting Point of Osazone, Degrees C.	Solubility of Osazone in Hot Water
A. J.	0.962	Glucosazone	205.6	Not perceptably soluble
T. P.	1.1	Glucosazone	203.8	Not perceptably soluble
W. A.	1.3	Glucosazone	204.0	Not perceptably soluble
T. J.	0.873	Glucosazone	204.6	Not perceptably soluble
O. M.	1.6	Glucosazone	205.0	Not perceptably soluble
J. W.	1.25	Glucosazone	206.2	Not perceptably soluble
W. S.	0.92	Glucosazone	206.0	Not perceptably soluble
O. W.	1.4	Glucosazone	205.2	Not perceptably soluble
F. A.	0.775	Glucosazone	205.8	Not perceptably soluble
A. L.	0.18	Glucosazone	204.4	Not perceptably soluble

The fact that the main portion of the reducing substance is fermentable indicates that it is glucose or galactose. The speed with which fermentation occurs points to the former. Galactose is fermented by yeast but more slowly than glucose. Definite data bearing on this

question were obtained in six cases. In these the urine contained more than 1 per cent. of reducing substance calculated as glucose. Quantitative determinations by means of Bertrand's method were compared with the rotation of polarized light. The values always corresponded much more closely with the specific rotation of glucose (52.5) than with galactose (81). For example: a specimen of urine gave a copper precipitate which was equivalent to 2.12 per cent. glucose. Calculated as galactose the percentage was 2.14 per cent. In a 2 decimeter polarimeter tube the rotation was 2.02 degrees, which calculated as glucose was equivalent to 1.92 per cent. sugar. Calculated as galactose the percentage was 1.24.

TABLE 4.—Influence on Urine Sugar of Change of Diet from Protein Milk to Water. Urine Collected for Period of 8 Hours and Sugar Output for 24 Hours Calculated from This

Name	Protein Milk	Water
	Sugar Output, Mg. per 24 Hours Calculated as Glucose	Sugar Output, Mg. per 24 Hours Calculated as Glucose
Gerald	640*	128*
Ellwood	506*	420*
Miller	610†	615†
Geyer	395*	340*
Cerecle	820*	195*
Kroph	560†	470†
Ratchfod	718†	230†
McLean	483†	406†
O'Neil	540*	204*
Lunetti	762†	607†
Smith	491†	602†
Peny	480*	163*
Barrows	623*	201*
Kintas	380*	350*
Miller	963*	770*
Sweney	820	695*

* Determined by micro-method of Benedict and Osterberg.
† Determined by the quantitative copper solution of Benedict.

The results of these determinations also offer additional evidence that the reducing agent was not lactose. Using the example cited, if the reducing action of the urine was due to lactose, the percentage would have been about 2.9. Calculated by the rotation, the percentage of lactose would have been 1.29. The mucic acid test was applied to these urines with uniformly negative results.

In addition to the chemical evidence there are a number of clinical observations which have a direct bearing on the nature of the reducing substance. If this substance is lactose or galactose it should be present in the urine only when lactose is ingested. This is not the case. Six infants with severe nutritional derangement (intestinal intoxication), whose urine showed marked reducing action, had received water or barley water alone for from sixteen to twenty-four hours.

Sixteen infants whose urine exerted a marked reducing action on copper solutions while receiving protein milk (Finkelstein's formula) as food were given water alone for from ten to eighteen hours. In six cases the reducing power of the urine was markedly decreased. In ten cases a quantitative determination showed only slight diminution of the reducing action of the urine (Table 4).

In consideration of these clinical tests alone, it seems inconceivable that the reducing action of the urine was due to lactose. No lactose was ingested and so far as is known the infant organism is incapable of storing or synthetizing lactose. On the basis of such evidence it seems that the reducing agent could only be glucose, the sugar of the body fluids.

While the results cited seem to warrant the conclusion that the reducing action of the urine of infants with nutritional disorders of the type observed is due to glucose and not lactose, it is not my intention to imply that alimentary lactosuria does not occur. Most of the patients on whom my investigations were conducted received food containing little lactose. This may perhaps explain to some degree the difference in my results and those of Grosz, Orban, Langstein and Steinitz and others.

In my experience, lactosuria has occurred only in infants who were not thriving and who were ingesting considerable lactose. The following case will serve as an example.

REPORT OF CASE

Baby F., aged 3 months, received in twenty-four hours 28 ounces of a milk and whey formula containing about 12.5 per cent. lactose. Just preceding a nutritional disturbance, the urine reduced Benedict's qualitative reagent, and a quantitative determination by Bertrand's method showed a reducing power equivalent to 1.95 per cent. glucose. Incubation with yeast for twenty-four hours showed no gas formation, and a second quantitative determination showed only a slight diminution of reducing power, a glucose equivalent of 1.58 per cent. After hydrolysis with 1 per cent. hydrochloric acid incubation with yeast for twenty-four hours caused distinct gas formation and a marked impairment of reducing action. Quantitative determination after fermentation for thirty hours showed an equivalent of about 0.3 per cent. glucose. The osazone had the appearance of lactosazone and dissolved almost completely in hot water.

These results would seem to indicate that the greater portion of the reducing substance was lactose although there was also in all probability a small amount of glucose. Similar results were obtained in the case of six other infants who were receiving moderate or large amounts of lactose in their food.

During investigations of lactose tolerance, lactosuria occurred in three cases. These cases, however, were exceptional. As a rule, when the urine reduced Benedict's qualitative reagent following the ingestion

of large amounts of lactose it was due to a readily fermentable sugar which was apparently glucose.

Infants with severe nutritional disorders usually secrete much less urine than normal which in consequence is concentrated. The question naturally arises whether the reduction of copper solutions by the urines of these patients actually represents an increased sugar output. Since normal urine always contains reducing sugar, it seems possible that with diminished urine secretion the concentration of sugar may be sufficient to reduce copper solutions without there being an actual increase in the amount of sugar eliminated. There seems little question that the increased concentration of the urine may be a factor in the frequency with which such urines reduce copper test solutions. If this were the only factor concerned, there might be only an increase in the percentage of urine sugar but no true glycuresis.

TABLE 5.—Comparison of Sugar Output in Urine During Period of Acute Symptoms and After Recovery. Urine Collected for a Period of 8 Hours and the 24-Hour Output Calculated from This

Name	Period of Acute Symptoms		After Recovery	
	Protein Milk, Amount Ingested in 24 Hours, Ounces	Sugar per 24 Hours as Glucose, Mg.	Food, Ounces	Sugar per 24 Hours as Glucose, Mg.
Ellwood	9	506	Milk 11 Lactose ½	206
Cerecle	7	820	Breast milk. 14	180
Lunetti	16	762	Protein milk 23 Maltose 1	207
Kinetas	2	380	Milk 14 Lactose ½	240
Miller	8	963	Protein milk 13 Cane sugar.. ½	193

Evidence on this question is given by quantitative determination of the total sugar output. Such determinations were made on the urine voided during eight hours, and the twenty-four hour elimination was calculated from this period. The total sugar per twenty-four hours calculated in this manner ranged from 280 to 963 mg. Some of the results are shown in Table 4. These figures indicate a greatly increased sugar elimination as normal infants on such a restricted sugar intake rarely put out more than 150 mg. Sugar eliminations comparable to these are found in normal infants only when very large amounts of sugar are ingested. Additional evidence was obtained in five cases in which the sugar output during the period of acute symptoms was compared with that after recovery. Despite the much greater sugar ingestion after recovery the sugar output was much less (Table 5). It,

therefore, seems evident that infants with nutritional disorders may show a true glycuresis.

Finally, the origin and significance of the increased sugar output must be considered. Is it due to a disturbance of sugar metabolism or is it merely of renal origin? Blood sugar determinations have been made in sixteen cases, and in all but two there was distinct hyperglycemia. The blood sugar after four hours' fasting ranged from 140 to 210 mg. per 100 c.c. This would seem to indicate a disturbance in the metabolism of sugar rather than the presence of a purely renal glycuresis. Increased blood sugar in the presence of increased urine sugar is generally assumed to indicate a disturbance in sugar metabolism.

As to the ultimate cause of the glycuresis, there is no direct evidence on which to base a definite opinion. There are a number of general observations on sugar metabolism which might have a bearing on this particular subject and which open a tempting field for discussion. It is my belief, however, that such discussion would go but a little way toward elucidating the problem and should be deferred until evidence bearing directly on the subject is available.

A PECULIAR FEVER OF INFANCY, PROBABLY DUE TO DEPLETION OF THE WATER RESERVE OF THE BODY*

C. G. GRULEE, M.D., AND B. E. BONAR, M.D.
CHICAGO

The following observations were made at a time when we were very much interested in the subject of fever in infants, which we were studying from another standpoint. The idea advanced by Balcar, Sansum and Woodyatt[1] that there might be a direct connection between a reduction of the water content of the body and the occurrence of fever is one which is especially attractive and probably can best be demonstrated, if at all, in the period of infancy. Most of the support from the literature which the theory has obtained has come from observations made on infants. It has, therefore, seemed to us that a report of some of our cases might be of interest.

REPORT OF CASES

CASE 1 (No. 132697).—George G., aged 2 months, entered the Presbyterian Hospital Feb. 15, 1920. The complaint was vomiting and undernourishment.

History.—The child was born by forceps delivery with a birth weight of 7¾ pounds. Pregnancy had been normal. He was put to the breast but did not nurse well and was fed breast milk with a spoon for some days. After two weeks he was given a mixture of 9 ounces of certified milk, 6 ounces of water, 2 teaspoonfuls of dextrimaltose and 20 drops of milk of magnesia. This was later increased to 10 ounces of milk, 18 ounces of water, 1 level teaspoonful of dextrimaltose, and 4 teaspoonfuls of milk of magnesia. For four days previous to entrance he had been given Horlick's malted milk. The day before entrance certified milk was again tried.

The child had never regained its birth weight and vomited practically everything. The type of vomiting was almost projectile. Vomiting became worse. The bowels had been regular, urination regular, the child slept soundly and was good natured. The bowel movements at times had been full of curds and sometimes were loose. There had been no convulsions or spasms.

Family History.—The family history was negative. This was the only child; there had been no other pregnancies. The father and mother were alive and well.

Physical Examination.—On physical examination there was found to be a poorly nourished infant who gave the impression of being very sick. The head was rather large; the fontanel was open; a slight nasal discharge was present; the face was pale and rather emaciated; no skin lesions were noted. The cervical and auricular glands were palpable; the neck was held back but no spasticity was noticed. Lungs and heart were negative; abdomen was distended. No peristalsis was noted at this time, fifteen minutes after taking nourishment,

* Received for publication, Aug. 15, 1920.

1. Arch. Int. Med. **24**:116 (July) 1919.

although the stomach was well outlined and there was some movement, but this was not a distinct peristalsis.

February 16, the von Pirquet reaction was negative.

February 17, the child vomited most of its food but seemed to retain enough not to lose weight.

February 18, the baby was rather constipated and still vomited.

February 19, peristaltic waves were seen passing over the stomach and there was projectile vomiting.

February 20, this peristalsis was much more marked as was also the vomiting which was definitely projectile.

Roentgen-ray examination indicated retention of food in the stomach; the stools were hard.

An exploratory laparotomy was done under local anesthesia but no tumor was found and the abdomen was closed.

February 21 and 22 the vomiting was still marked.

February 22, a continuous enema of 6 per cent. dextrose solution was given. This was continued through the twenty-third and the twenty-fourth.

February 25 the child began to retain food.

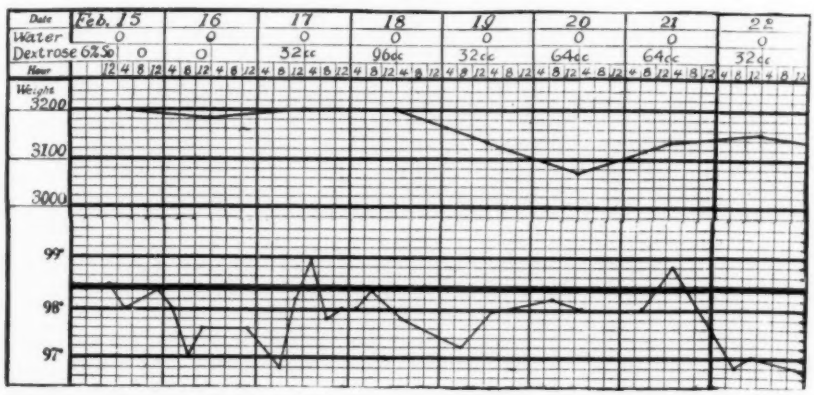

Fig. 1.—George G.; prefebrile period. Food: Albumin milk, 576 c.c.; dextrimaltose, 24 gm. Six feedings of 96 c.c. each.

February 28, 55 c.c. of a 6 per cent. solution of dextrose was given subcutaneously.

Clinical Course.—The course of this case so far as temperature was concerned may be divided into the prefebrile period, the febrile period, and the postfebrile period. The prefebrile period extended from February 15 to February 23. During this time the child obtained as food 18 ounces (576 c.c.) albumin milk; ¾ ounce (24 gm.) dextrimaltose, six feedings of 3 ounces (96 c.c.) each. During this period there was no rise in temperature. The child received 6 per cent. dextrose solution, three times a day, beginning with the seventeenth. The child obtained from 1 to 5 ounces a day of this solution (32 to 160 c.c.). February 18, atropin sulphate, $\frac{1}{1,000}$ grain, was administered hypodermically every eight hours. This was discontinued on the twenty-third. The stomach was washed before each feeding; this, too, was discontinued on the twenty-third.

The second period is the febrile period, which lasted from February 23 to March 7. During this time the child received food consisting of 8 ounces (256 c.c.) whole milk, ¾ ounce (24 gm.) dextrimaltose, 1 ounce (32 gm.) flourball, to which 8 ounces (256 c.c.) of water was added and the whole boiled

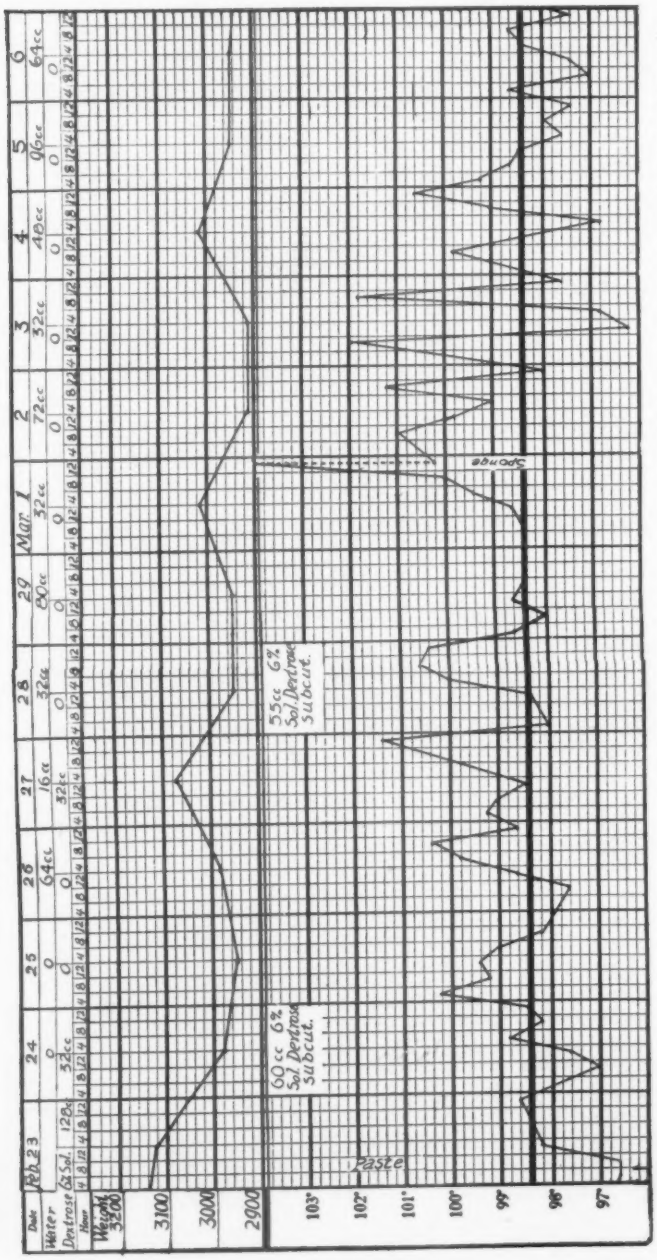

Fig. 2.—George G.; febrile period. Food: Paste made from whole milk, 256 c.c.; dextrimaltose, 24 gm.; flourball, 32 gm.; water, 256 c.c.

down to a thick paste. Atropin was given, 1/1,000 grain, every four hours; strychnin, 1/1,000 grain, three times a day, both being given hypodermically. An attempt was made to give dextrose solution by the drop method rectally but without much success because the child retained very little of it. The strychnin was discontinued on the third, and on that day the atropin was ordered given only twice a day. Only small quantities of water were given at this time, varying in twenty-four hours from 16 to 96 c.c. February 28, the child was given 55 c.c. of 6 per cent. dextrose solution subcutaneously.

The third or postfebrile period began March 7 and continued through to the twenty-third. During this period the paste was gradually replaced, one feeding at a time, with liquid, consisting of 2 ounces (64 c.c.) milk, and ¼ ounce (8 gm.) dextrimaltose. The first of these feedings was given March 7. March 15 two feedings were given; on the seventeenth three feedings, and from the nineteenth on all feedings were of liquid nourishment.

During this last period the temperature exceeded 99 F. three times. On the afternoon of March 8, after the child had shown a subnormal temperature, dropping following liquid nourishment, the temperature rose to 100.8 F., dropping immediately to 96.6 F. On the following afternoon, March 9, the temperature rose to 99.4 F., and on the afternoon of the twentieth the temperature at one time registered 99.6 F. With these exceptions, this period was in marked contrast to the period when the child received only a thickened paste. Throughout the treatment the child refused water, the total daily quantity never exceeding ½ ounce (16 c.c.). The urine was negative on several examinations. February 16, the blood showed: Erythrocytes, 4,500,000; leukocytes, 7,500; hemoglobin, 75 per cent. Differential count: Small lymphocytes, 56 per cent.; polymorphonuclears, 44 per cent.

Comment.—Practically the only difference between the second period and the first and third periods consisted in the fact that the quantity of water which the child took was very much reduced in the former. There was no other circumstance which was clinically evident which we could in any way connect with a rise in temperature. It should be remembered that this was an extremely emaciated infant, as the weight curve shows, that it had been subjected to a long period of reduction by vomiting, and that, hence, in all probability the water content of the body was greatly reduced.

Case 2 (No. 133045).—Mary Z., aged 6 months, entered the Presbyterian Hospital, Feb. 27, 1920.

History.—Born spontaneously with a birth weight of about 7 pounds. Breast fed for five months during which time her general condition was good. One month before entrance, the child had a cold and a slight cough. During the daytime the child had been vomiting. Its food at the time of entrance had been four feedings of cow's milk, three parts milk to one of water, with two teaspoonfuls of sugar per bottle, in addition to which had been given one cereal and one vegetable feeding. The child was apparently doing well, although there was a history of some vomiting. Two days before entrance the mother had noticed some discharge from the ear. Previous to this time the child had had no disturbance of any sort. Mother stated she was easy to take care of, took her food eagerly and slept well.

Family History.—This was the sixth child, the oldest being 14 years of age. Two miscarriages; children all apparently well; father alive and well. The mother soon after the entrance of the child into the hospital was taken to an insane asylum because of dementia praecox.

Physical Examination.—This revealed nothing abnormal, except that the child was poorly nourished, weight about 10 pounds. There was a somewhat

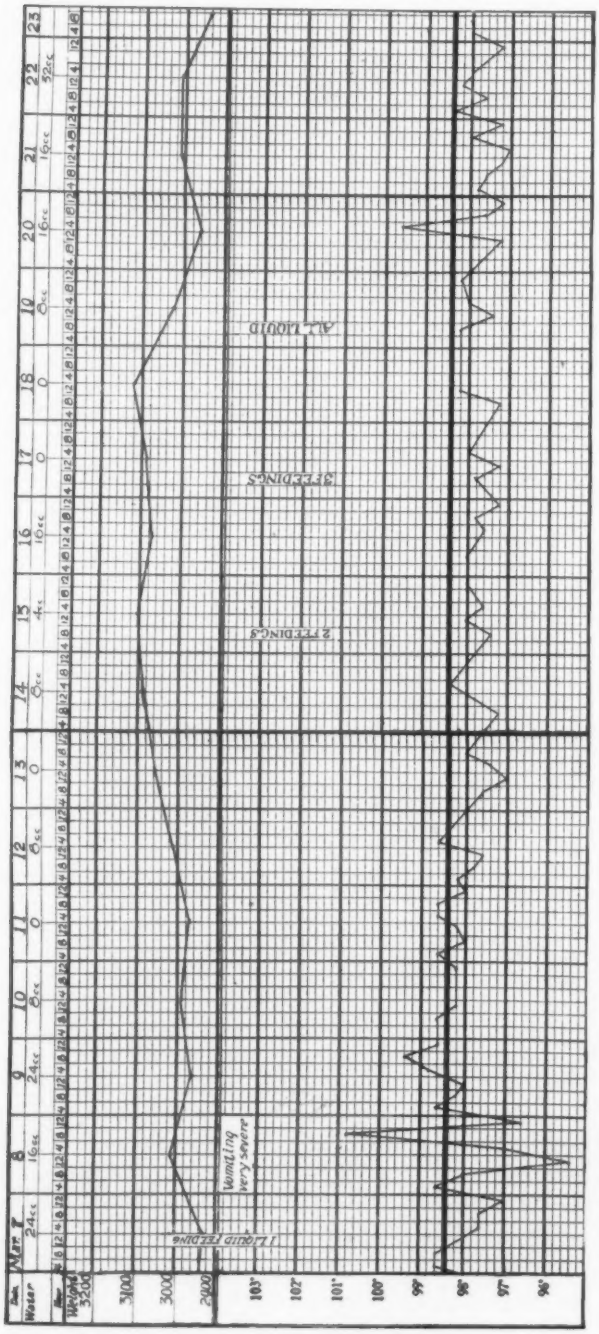

Fig. 3.—George G.; postfebrile period. Food: Each liquid feeding consists of whole milk, 64 c.c.; dextrimaltose, 8 gm.

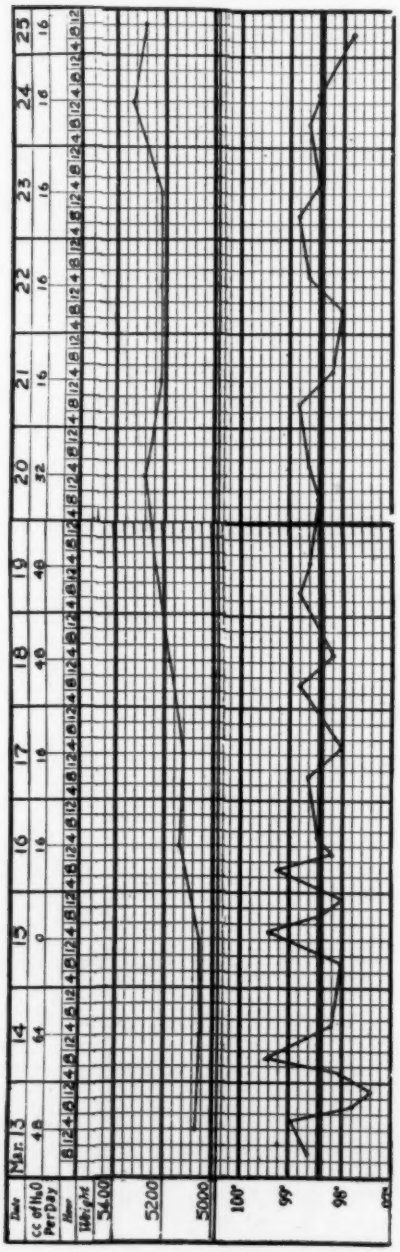

Fig. 4.—Mary Z. First period. Food, March 13: Albumin milk, 1,056 gm.; dextrimaltose, 32 gm. Five feedings of 224 c.c. each.

purulent discharge from the right external auditory meatus. Other physical findings were negative.

The discharge from the ear ceased March 3 and never recurred thereafter. During the early days of the child's stay in the hospital its food consisted of 30 ounces (960 c.c.) albumin milk; 1 ounce (32 gm.) dextrimaltose, five feedings of 6 ounces (192 c.c.) each. This was increased to 33 ounces (1,056 c.c.) albumin milk; 1 ounce (32 gm.) dextrimaltose, five feedings of 7 ounces (224 c.c.) each on the ninth. Although the child had been vomiting more or less throughout its stay in the hospital, rumination was first actually noticed March 22, and the food was not changed until March 25.

Clinical Course.—From the time of its entrance to March 25 constitutes the first period in our history. During this period the temperature was nearly always within normal limits. March 2 it rose once to 99.4 F.; March 10 it rose once to 100 F.; March 11 it rose once to 99.6 F., and once to 99.2 F. March 14 the temperature was 99.6 F. once; March 15, 99.6 F. once; March 16, 99.2 F. With these exceptions, the temperature from February 27 to March 25, in all twenty-eight days, never rose above 99 F. During this first period, in addition to the food, the child obtained some water between feedings.

As medication during this first period, from March 13 to March 24, the child received a teaspoonful of cod liver oil and phosphorus twice a day. March 24 this was increased to three times a day and remained the same throughout the course. March 24 the child was given $\frac{1}{1,000}$ grain atropin hypodermically, every four hours, and this was kept up for two days when the dose was reduced to $\frac{1}{2,000}$ grain and this was continued until March 30.

The second period began March 25. The child was given food consisting of 15 ounces (480 c.c.) whole milk, ½ ounce (16 gm.) dextrimaltose, 1½ ounces (48 gm.) cream of wheat, the whole boiled down to a paste and given in five feedings. March 26 this was increased to 18 ounces (576 c.c.) milk, ½ ounce (16 gm.) dextrimaltose, and 2 ounces (64 gm.) cream of wheat. March 28 this was still further increased to 20 ounces (640 c.c.) whole milk, ¾ ounce (24 gm.) dextrimaltose and 2 ounces (64 gm.) cream of wheat. This latter feeding was continued until May 3.

Beginning the afternoon of March 26, the temperature became irregular and remained so until the afternoon of May 3. During this time the quantity of water consumed was very low. The only days when the temperature was within normal limits were April 3 and 4. On these two days the quantity of water given was not in excess of that consumed in the other period.

The third period began May 3. At that time the child was given 20 ounces (640 c.c.) milk, ¾ ounce (24 gm.) dextrimaltose, 20 ounces (640 c.c.) water, five feedings of 8 ounces (256 c.c.) each. On the afternoon of May 3 the temperature showed a sudden drop to normal. May 4 in the afternoon the temperature dropped to 95.2 F. and the child was in a state of collapse. The Intern became alarmed and ordered that the child be put again on thick cereal feeding. The condition was so alarming that the child was given camphorated oil, 8 minims, and strychnin sulphate, $\frac{1}{500}$ grain, hypodermically. This was continued for two further doses and early in the morning of May 5 it was necessary again to give the child 8 minims of camphorated oil. When the child was seen by the attending man next morning, the liquid food was again ordered and by noon the temperature had dropped to 97.8 F., and in the afternoon it fell to 96.4 F.

This feeding was continued until May 10. The temperature became markedly subnormal; on the eighth it rose to 99.4 F., then dropped to 96 F.; on the ninth, it dropped to 96 F.; the morning of the tenth it again rose to 99.4 F., and dropped to normal that afternoon.

In the fourth period, beginning May 10, the child was again put on the paste consisting of 20 ounces (640 c.c.) whole milk, ¾ ounce (24 gm.) dextrimaltose, 1 ounce (32 gm.) cereal in five feedings. This was continued until

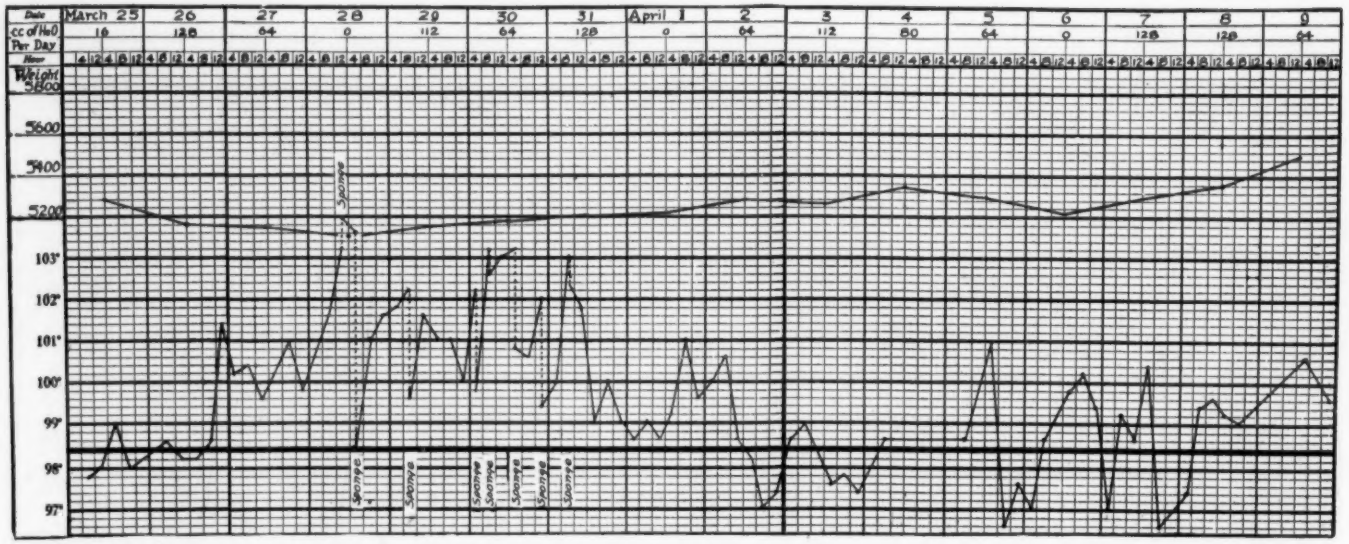

Fig. 5.—Mary Z. Second period. Food, March 25: Paste consisting of whole milk, 480 c.c.; dextrimaltose, 16 gm.; cream of wheat, 48 gm. March 26-28: Paste consisting of whole milk, 576 c.c.; dextrimaltose, 16 gm.; cream of wheat, 64 gm. March 28 to May 3: Paste consisting of whole milk, 640 c.c.; dextrimaltose, 24 gm.; cream of wheat, 64 gm.

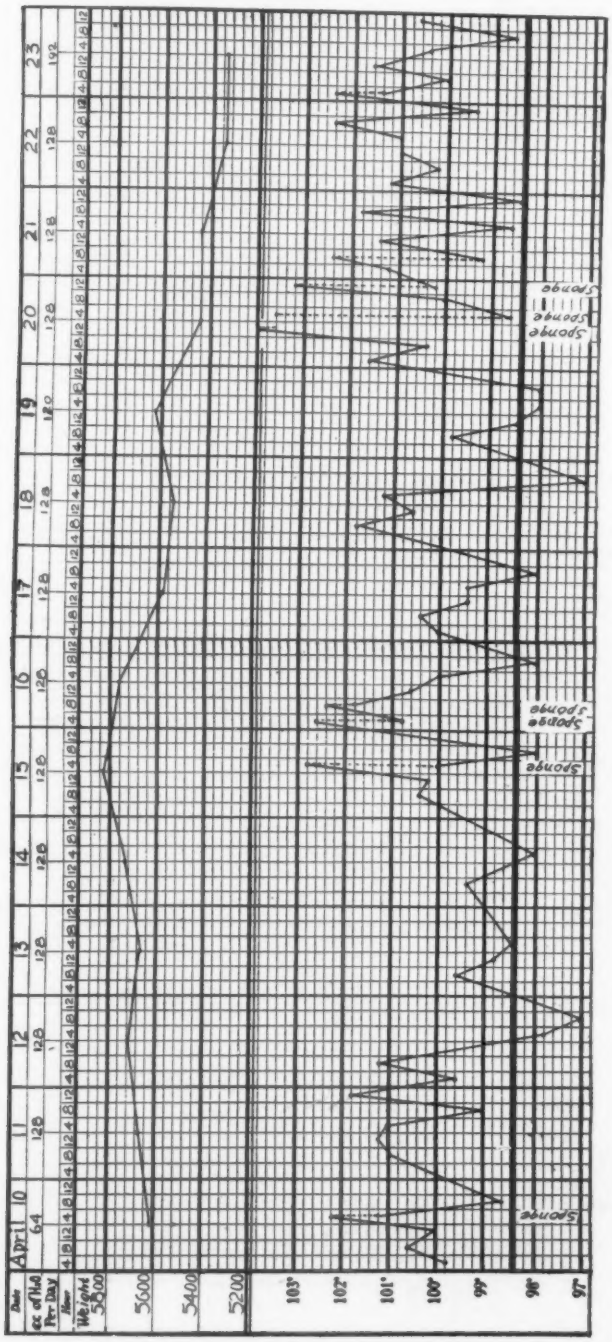

Fig. 6.—Mary Z. Second period (continued). Food: Same.

the fifteenth. During this period the child again showed a markedly febrile temperature.

May 15, when the fifth period began, the child was put on food exactly the same as during the previous period (fourth), except that 20 ounces (640 c.c.) water was added and the whole was not made into a paste, and five feedings of 8 ounces (256 c.c.) each were given. The afternoon of May 15, the child again showed a marked drop in temperature, 96.4 F. This was followed by a slight rise to 100.6 F. the next day. The child was perspiring very freely. The temperature dropped to 96.4 F. on the seventeenth and remained low.

May 25 the food was again changed (sixth period), the same food being given as during the fourth period. The afternoon of May 25, the temperature rose to 101 F. and on the twenty-sixth it rose to 101.6 F. It was decided then to push water, and the quantity, which for the previous ten days had varied between 32 and 96 c.c. per day, was increased so that the following quantities were given:

May 26	304 c.c.	May 31	384 c.c.
May 27	400 c.c.	June 1	328 c.c.
May 28	208 c.c.	June 2	320 c.c.
May 29	308 c.c.	June 3	448 c.c.
May 30	288 c.c.		

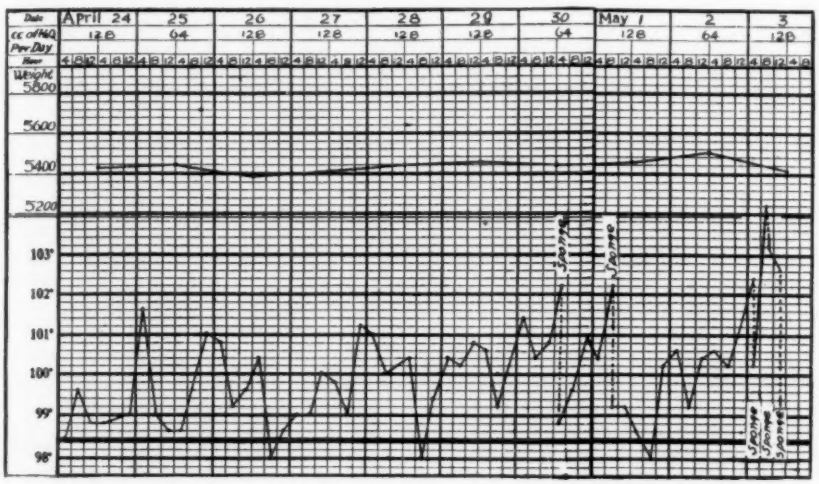

Fig. 7.—Mary Z. Second period (continued). Food: Same.

The afternoon of the twenty-sixth, the temperature dropped to normal; on the twenty-seventh it was subnormal; on the twenty-eighth it was normal. It was slightly above normal on the twenty-ninth, and the afternoon of the thirtieth it began to rise. From then on until June 3, the temperature remained somewhat high, though lower than in the previous period.

June 3 (seventh period), the food was again changed back to that of the fifth period. The temperature on the fourth dropped to 95.2 F. and remained subnormal with one exception until the fourteenth. On the ninth the temperature rose once to 101 F. This was a hot, humid day.

The quantity of water consumed during this period varied between 64 and 272 c.c.

During the next period (the eighth) from June 14 to June 25, the same food was given as in the sixth period. June 15, the temperature rose to 101 F., but with the administration of water, 400 c.c. June 16, 336 c.c., and the seventeenth, 360 c.c. water were given and the temperature remained

down. Water was restricted on the seventeenth so that the child after that received for this period no more than 96 c.c. per day, with the result, that the remaining part of this period the child ran a febrile temperature throughout.

June 24 (ninth period), the child was again put on a liquid food of the same composition as during the seventh period. That evening, the temperature dropped to 97.2 F., remaining subnormal for three days thereafter, being within normal limits until the end of this period, July 2.

Comment.—The difference in temperature bore such direct relation to the water intake throughout this case that we decided to see if it were possible to obtain any confirming evidence by blood examination. For this purpose there were made careful estimates of the number of red and white cells, the hemoglobin, and the specific gravity. The specific gravity was estimated by the benzol-chloroform method, the specific gravity of the liquid being carefully estimated by means of a Westphal balance.

Blood Examination.—The blood examination in each instance was made at 10 a. m., one hour after the ingestion of food.

TABLE 1.—BLOOD FINDINGS IN CASE OF MARY Z. DURING SIXTH PERIOD WHILE THE CHILD WAS ON LIQUID NOURISHMENT

Date	Hemoglobin	Erythrocytes	Leukocytes	Sp. Gr. at 16 C.
6/10/20	85%	4,790,000	6,700	1.056
6/11/20	85%	4,864,000	7,650	1.052
6/12/20	90%	4,998,000	8,600	1.062

TABLE 2.—BLOOD FINDINGS IN CASE OF MARY Z. DURING THE SEVENTH PERIOD WHEN THE CHILD WAS ON FOOD PASTE

Date	Hemoglobin	Erythrocytes	Leukocytes	Sp. Gr. at 16 C.
6/19/20	95%	5,300,000	9,980	1.072
6/20/20	92%	5,460,000	9,700	1.069
6/21/20	95%	5,330,000	9,000	1.070

Comment.—If we may regard the recent observations of Wimberger [2] as conclusive, this observation of the difference in the concentration of the blood is quite remarkable in that in his observation on children between 7 months and 17 years of age, he found that the variation in the volume of the blood serum was extremely small, and he thinks that the blood is independent of the water content of the surrounding tissues, even in very marked changes in weight.

When we stop to consider the cause of fever in these cases, we naturally revert to the various theories which have been advanced as the reason for the increase of temperature in the human body. These may be placed into three groups: (1) Fever due to some involvement of the central nervous system; (2) fever of an infectious or bacterial origin, and, (3) dehydration fever. In neither of the cases here

2. Ztschr. f. Kinderh. **25**:64, 1920.

reported need we consider the question of fever caused by some derangement of the central nervous system. There was certainly nothing of that nature in either of the cases. Therefore, we must be confined in our discussion to the last two causes; namely, bacterial and dehydration.

The question that arises in both these cases is whether or not the changed conditions from the removal of water from the food are responsible for an absorption of bacteria from the intestine with a resulting bacterial infection and toxemia. We may certainly dismiss from our discussion any other source of bacterial infection, and we

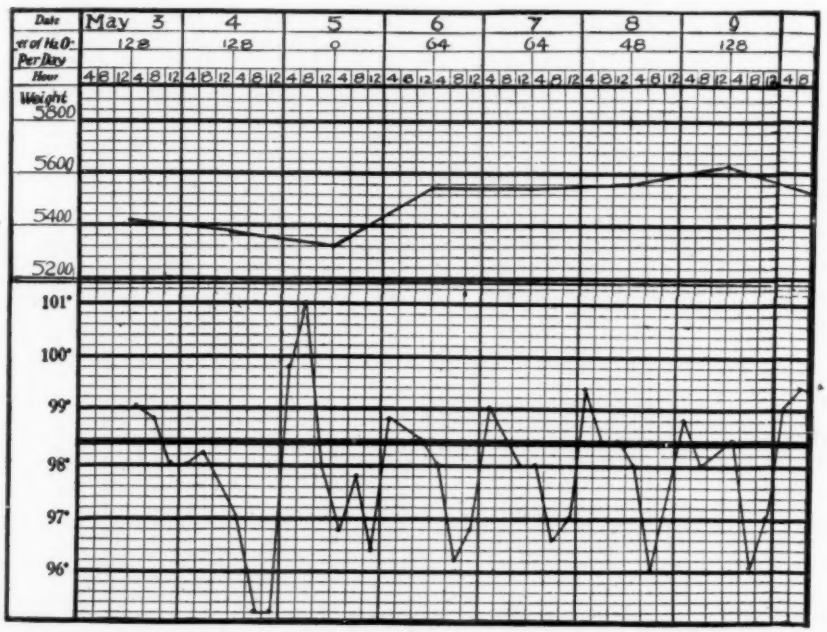

Fig. 8.—Mary Z. Third period. Food: Whole milk, 640 c.c.; water, 640 c.c.; dextrimaltose, 84 gm. Five feedings of 256 c.c. each.

think it fair to assume that there is a very definite connection between the giving of thickened food and the rise of temperature, since in both cases there is evident such a definite time relation between the two conditions, the temperature falling quite evidently as the result of the giving of liquid food. While it would be impossible to state absolutely that there was no bacterial infection, or absorption of dead bacterial bodies under the circumstances above mentioned, still there are many points which speak very decidedly against such a condition. In the first place, although the temperature curve, especially in Case 2, lasted over many weeks, there was at no time any evidence of

toxemia. The child was somewhat disturbed by thirst, but no general symptoms of toxemia or septicemia such as are regarded as characteristic of these conditions were to be observed in the child. In the second place, if such infectious conditions did occur, we would certainly expect that there would be shown some evidence of involvement of the gastro-intestinal tract. In conditions due to bacterial infection from the gastro-intestinal tract, or to toxemia produced by conditions within the gastro-intestinal tract, the gastro-intestinal symptoms are

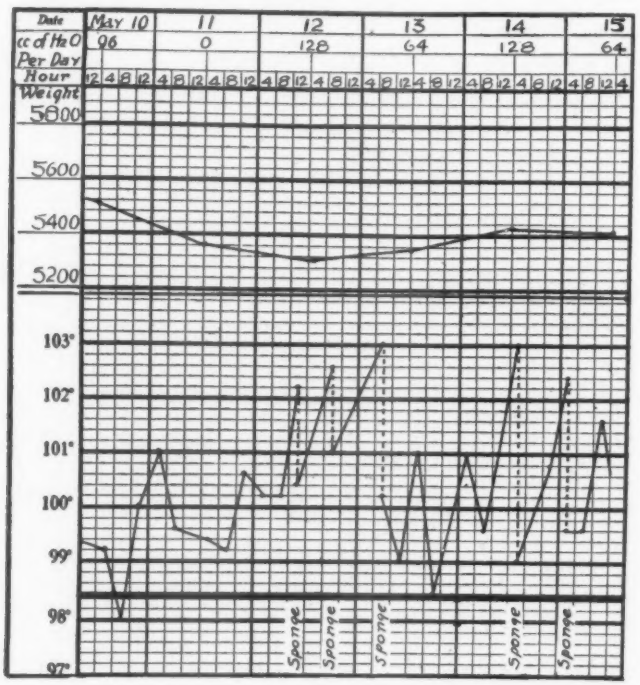

Fig. 9.—Mary Z. Fourth period. Food: Paste consisting of whole milk, 640 c.c.; dextrimaltose, 24 gm.; cream of wheat, 32 gm.

practically always in proportion to the height of the temperature. In other words, when temperature elevation depends on a gastro-intestinal disturbance, the number of stools is increased and the character of the stools is changed to a much greater degree than if that same temperature is produced by an infectious process which does not refer to the gastro-intestinal tract.

Diarrhea was not present in either of these cases at any time, nor was there any perceptible change in the character of the stool, no matter whether the child was on the liquid or the solid food. We may, therefore, say that while we cannot absolutely rule out bacterial

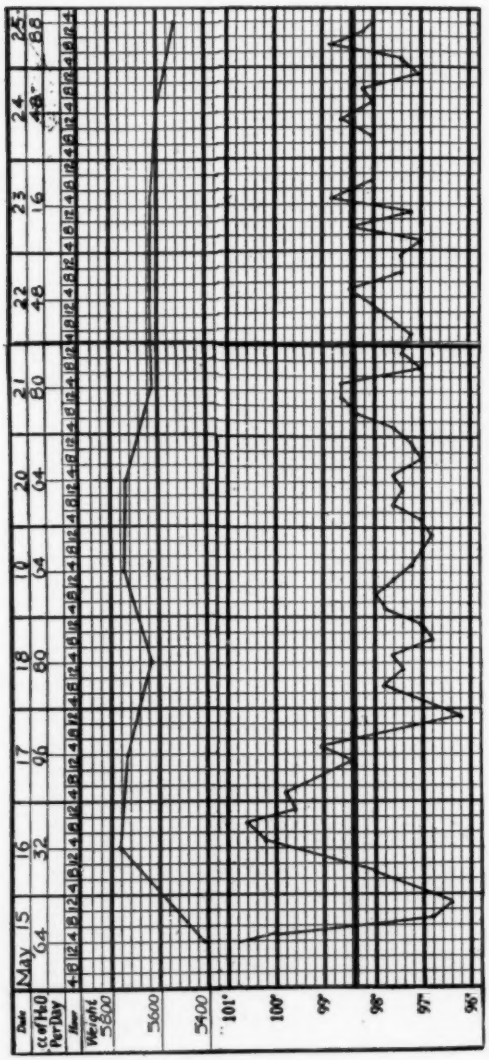

Fig. 10.—Mary Z. Fifth period. Food: Whole milk, 640 c.c.; dextrimaltose, 24 gm.; cream of wheat, 32 gm.; water, 640 c. Five feedings of 25c.c. each.

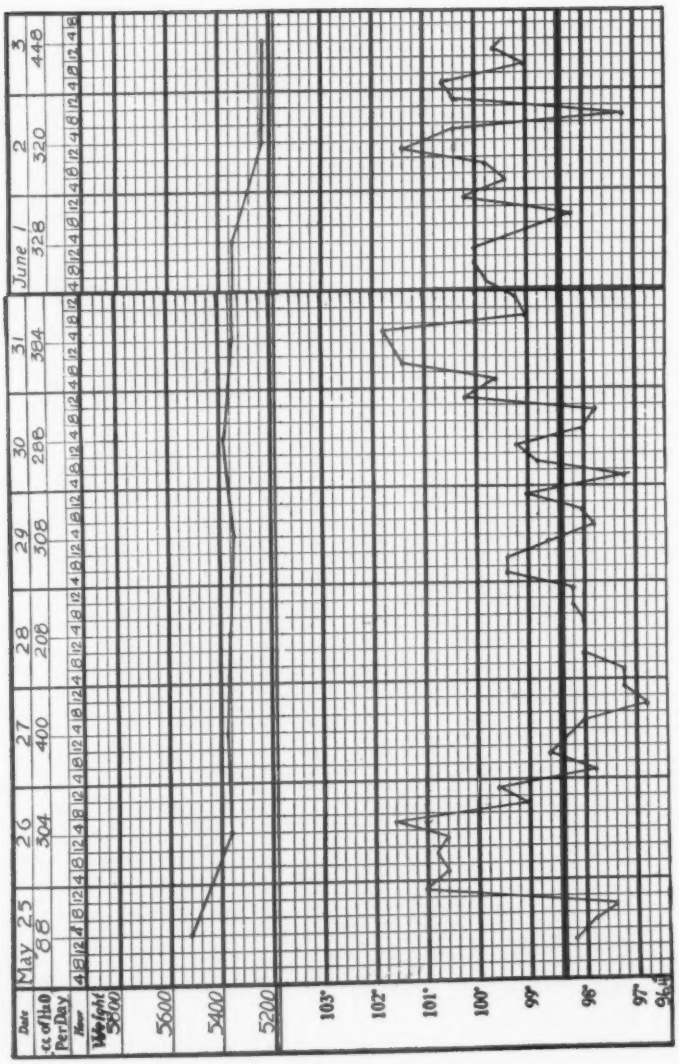

Fig. 11.—Mary Z. Sixth period. Food: Same as in fourth period.

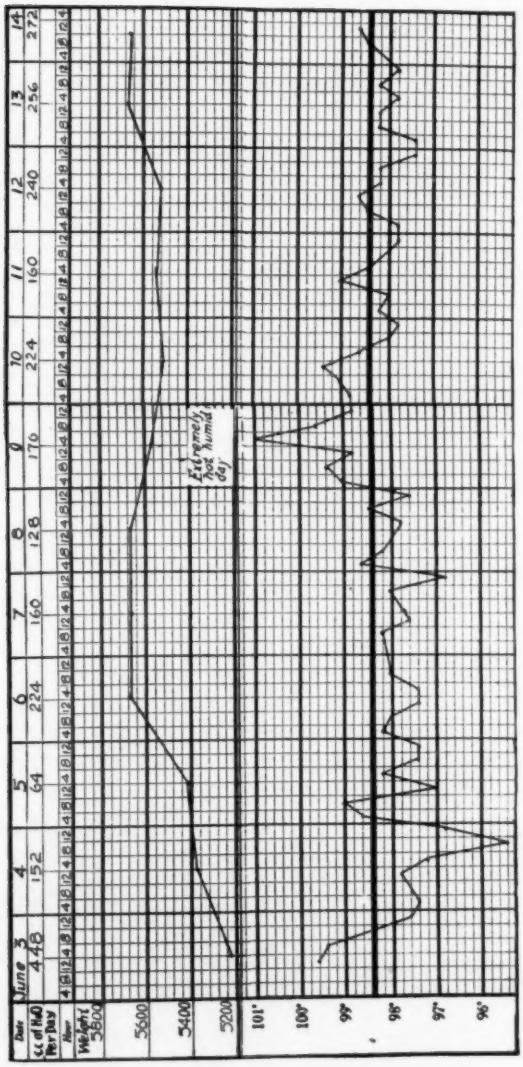

Fig. 12.—Mary Z. Seventh period. Food: Same as in fifth period.

infection in this instance, all clinical signs point very strongly against such an element entering into the explanation of this fever.

In considering the third cause of fever, dehydration, many points in both these cases, but especially in Case 2, favor such an explanation. First, there is the very definite increase of temperature within a short time after the beginning of the thick paste feeding. Second, there is the continuation of this fever throughout the entire time when the paste is being given. Third, this fever ceases very shortly after the child is put on liquid food. Fourth, for a certain length of time while the child is on thickened food this fever may be reduced by the introduction of large quantities of water by mouth. Fifth, there is distinct evidence of a difference in the water content of the blood between the period of liquid feeding and that of thickened feeding, the blood being much more highly concentrated during the latter than during the former period.

It seems likely that the water content of the body exists really in three forms; available water, residual water and essential water. If we accept the dehydration theory of fever, and apply it to these cases, it would seem likely that in both cases, due not to malnutrition but to undernutrition caused by the rejection of food through vomiting and rumination, the system of each of these infants had lost any surplus which the body might have contained had these processes not been present. When the paste food was given, therefore, there was withdrawn from the body for the purposes of digestion a rather large quantity of water. The withdrawal of this water resulted in a dehydration, with consequent rise of temperature. When the digestive processes were completed, this water could again be absorbed, with the result that an up and down temperature curve would be produced. That the margin is very narrow may readily be seen by the quantity of water which is sufficient to reduce this temperature. A difference of from 150 to 200 c.c. a day was sufficient to produce a cessation of the fever, at least temporarily. In the first case, too, there was a very definite change soon after the first liquid feeding was given, although the quantity of fluid was very small.

One phenomenon occurred in the last case which we are not able to explain satisfactorily at present, and that is the acute collapse which occurred when liquid food was given following a period of thickened food. It is interesting to note that the collapse was most severe following the prolonged use of the thickened food, and was less marked when the thickened food had been used only a short time. There seemed, however, to be that tendency after each period of thickened food nourishment. The only clinical picture which we know of that corresponds to that seen in these cases is the acute collapse

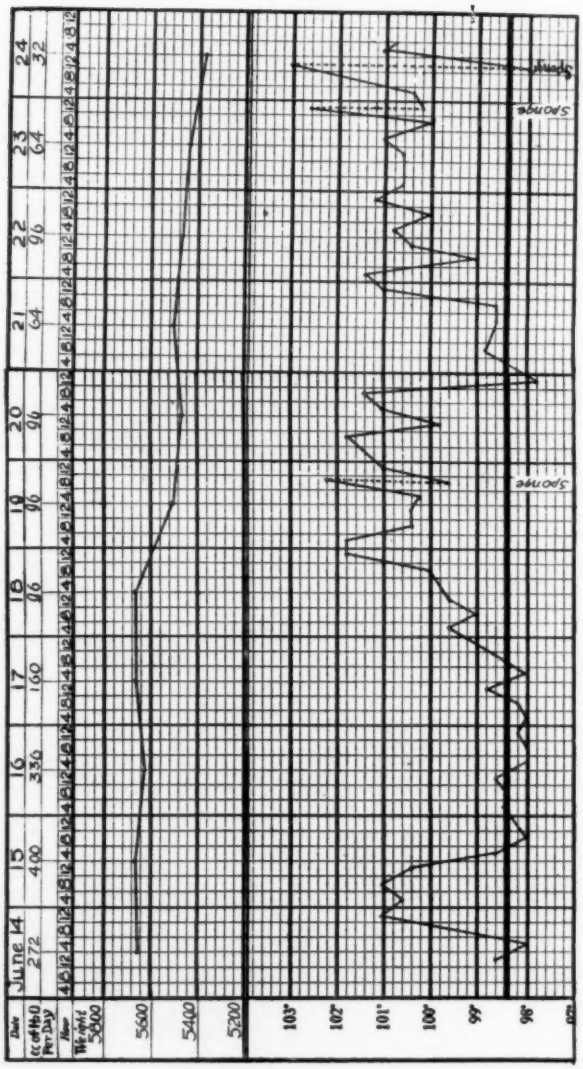

Fig. 13.—Mary Z. Eighth period. Food: Same as in sixth period.

sometimes seen in severely marantic infants. Whether these two conditions are identical or not it would be impossible to say.

The practical importance of these observations suggests itself at once. Fevers of unknown origin have always been described in infancy. There have also been described fevers whose explanation was certainly inadequate; such, for instance, as the temperatures which have been described during teething. Such fevers might possibly, if properly analyzed, fall into a group like that described in this paper. It should be noted, however, that such fevers are to be described only under certain conditions and those conditions must correspond to a dehydration of the organism. Under what conditions the organism is susceptible of showing fever following dehydration, would be hard to say, but it is very evident that one would expect to find such a state in those infants who had been depleted by severe vomiting.

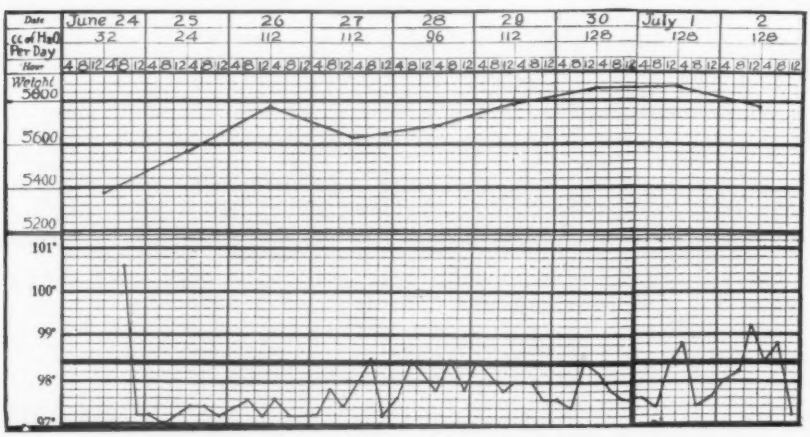

Fig. 14.—Mary Z. Ninth period. Food: Same as in fifth period.

Another practical point is raised as to whether or not the use of thickened food is justified in the treatment of severe vomiting conditions in infancy; such, especially, as pyloric stenosis, pylorospasm and rumination. In the first place, it should be emphasized that the food used in these cases which we have presented was not simply a gruel but was in reality a thick paste. The water content, therefore, was reduced to the minimum, more, in all probability, than would be true in the ordinary thickened feeding used for the type of case mentioned; but even though the temperature did develop, it would have little effect on the general economy of the child, and could easily be reduced by giving some liquid food. A note of warning, however, should be sounded in these cases as to the manner in which the food is changed. If such a temperature has previously existed, the change

from the thickened food to the liquid food should always be made very gradually. We know of at least one child that had pyloric stenosis which died under conditions which in the light of our recent experience could most easily be explained on the basis of an acute collapse following giving liquid food after thickened food was used.

In relation to water content of the body, it would seem that the margin is very narrow between sufficient quantity of water within the body to maintain normal temperature and that present during the febrile period. In other words, after the available water is removed a very slight further reduction will result in the production of temperature. It has been known for some time, and has further been substantiated by Wimberger, that the water available for use by the organism is much more readily obtained from the tissues than from the blood. The question in view of the findings in the last of these cases is raised whether we may regard the water content of the blood as indicative of the state of the organism in relation to the residual water. There was in this instance during the febrile period a very decided reduction in the water content of the blood, as shown by blood counts, hemoglobin estimation and specific gravity. If temperature elevation occurs only when such depletion of the water content of the blood takes place, then we would have a very definite standard by which to judge of the condition in future observations.

CONCLUSIONS

There occurs in infants who have been depleted by vomiting or rumination following the use of a thickened paste feeding, a temperature curve which can most easily be explained on the basis of dehydration, though it is not possible absolutely to rule out absorption of bacteria as a cause, either total or partial, of this temperature. This temperature is unaccompanied by toxic or gastro-intestinal symptoms and there is evidence of a reduction in the water content of the blood during the febrile period.

FEEBLEMINDEDNESS IN HEREDITARY NEUROSYPHILIS *

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BOSTON

The ever persistent effort of nature unassisted to return successive progeny more and more to a normal constitution by combating interceding infections and other adverse influences, is an old and well known fact, especially well known in the course of syphilis. In congenital syphilis this phenomenon has been observed and studied mainly from the somatic viewpoint. The marked improvement in physical condition of each succeeding child born to a syphilitic parent over the preceding one is a matter of every day obstetric experience. From observations on the psyche of congenital syphilitics, and a comparison of mental reactions in children nearer and farther removed from the source of infection, one is convinced that there is a similar progressive degradation of the syphilis toxin affecting the brain cells.

It has been the custom [1] in the neurosyphilis clinic of the Psychopathic Department of the Boston State Hospital and other psychopathic hospitals [2] to apply the Wassermann test to the blood serum of the children of patients found to be afflicted with one or another form of neurosyphilis. Solomon was able to show that 8.4 per cent. of children (in 160 families) returned a positive Wassermann reaction in the blood. Jeans,[3] in a study of 100 syphilitic families, found 78 per cent. of the living children to be syphilitic. The same author [4] reported on 214 seropositive infants and children with hereditary syphilis, and found sixty-six, or 38 per cent., with positive cerebrospinal fluid Wassermann reactions accompanied by gross mental defect in twelve. He also found, in a review of the literature, approximately 33 per cent. of cases with central nervous system involvement, as judged by spinal fluid findings.

An attempt is here made to determine the incidence of central nervous system invasion among this class of children. To this end lumbar punctures were performed and the cerebrospinal fluid subjected

* Received for publication September 14, 1920.

1. Solomon and Solomon: The Family of the Neurosyphilitic. Mental Hygiene **2**:71 (Jan.) 1918.

2. Barrett, A. M.: Mchigan State Psychopathic Hospital, Sixth Biennial Report, June 30, 1918, p. 61.

3. Jeans, P. C.: Am. J. Dis. Child. **11**:11 (Jan.) 1916.

4. Jeans, P. C.: Cerebrospinal Involvement in Hereditary Syphilis, Am. J. Dis. Child. **18**:173 (Sept.) 1919.

TABLE 1.— RÉSUMÉ OF PHYSICAL AND MENTAL DATA OF THIRTY CHILDREN

Case Number	Age, Years	Mental Age, Years	Wassermann Reaction		Globulin	Albumin	Cells	Colloidal Gold Test	Remarks
			Serum	Cerebro-spinal Fluid					
1	13	6 8/12	+	+	0	N	1	5555441000	
2	12	8 8/12	—	—	0	N	0	0000000000	(Family A)
3	9	7 4/12	+	—	0	N	1	0012100000	Father and mother paretic
4	8	4 10/12	+	—	0	N	0	0033310000	(Table 2)
5	4	3 8/12	—	—	0	N	10	0000000000	
6	16	..	—	..	..	..	..	.....	(Family B) Case 6, Normal
7	12	(8.2)	+	+	0	N	37	5555531000	Father syphilitic, WRS +, CSF —
8	6	3.5	+	—	0	N	1	1111210000	Mother WRS —
9	5	..	—	—	0	N	0	0000000000	(Table 3)
10	11	10 1/5	+	—	0	N	2	0000100000	
11	11	8 11/12	+	—	0	N	1	Negative	Truant; backward at school
12	11 2/12	9 2/12	+	—	0	N	2	Negative	
13	11	2 to 3 (est.)	..	..	..	..	..	.....	Imbecile; deaf-mute
14	11 5/12	10 1/2	+	—	0	N	0	Negative	
15	10	Backward in school	+	—	0	N	6	0000100000	(Family C)
16	8	..	+	—	0	N	1	0001000000	Father paretic
17	6	..	..	..	..	..	..	.....	Mother WRS —, WRFl —
18	9 1/2	8.1	+	—	0	N	0	Negative	(Family D)
19	4	..	—	..	..	..	..	.....	Father WRS ; WRFl —
20	15	..	+	—	0	N	0	Negative	(Family E)
21	9.3	11	+	—	0	N	0	Negative	Father died of general paresis
22	1 1/2	..	—	..	..	..	..	.....	No. 20 has iritis; mother WRS +
23	9	8.5	+	—	0	N	3	Negative	Father and mother, WRS +
24	6	..	+	+	2	2	26	5555441100	Hydrocephalus
25	4	..	+	—	0	N	1	Negative	
26	8 9/12	8 2/12	—	..	..	..	..	.....	(Family F)
27	7	..	+	—	0	N	0	Negative	Mother, WRS +
28	6 3/12	4 est.	+	+	+	+	?	1122100000	By test 2% uncooperative
29	3 5/12	2 3/5	—	..	..	..	..	.....	
30	2	..	—	..	..	..	..	.....	Saddle nose; high palate; double Babinski

to the usual analysis. In order to further study the effect of the syphilotoxin on the brain, psychologic tests have been employed. In families in which there are several living children showing variant degrees of syphilitic attrition, it appears that the mental and physical ratings bear a definite relation to each other.

The series of cases studied consisted of thirty children ranging from 2 to 16 years, in either one or both of whose parents there was a positive Wassermann reaction on the blood serum. Most of the parents had neurosyphilis, usually of the paretic type. In some instances, the family became known to the clinic through a congenitally syphilitic child, brought to the hospital on account of mental disease or mental defect (backward and special-class school children). In these families the diseased parent was usually free of syphilitic mental disease though always seropositive.

Results of examinations of blood serum or blood serum and spinal fluid (Wassermann reaction) are given in thirty children either one or both of whose parents were seropositive. Of these children, nineteen showed a definitely positive Wassermann reaction. With few exceptions, the older children, i. e., those born nearest the time of the parental infection, showed a positive Wassermann reaction; the younger and farthest removed usually showed a negative Wassermann reaction. When the infection occurred during a series of pregnancies, those children born before returned a negative Wasserman reaction; those born after, if living, showed marked evidence of hereditary syphilis; succeeding children showed decreasing evidence of it.

On twenty-two of the thirty children a spinal puncture was obtained, and in four of these fluids a positive Wassermann was returned. The colloidal gold test in three of these four positives was typical of paretic neurosyphilis. In the fourth case (No. 28) it was not; there had been some treatment (intravenous arsphenamin), though not enough to cause such a change, even if the fluid might be assumed to have previously been capable of a frank paretic gold curve.

Together with the last mentioned case there were three others (Cases 3, 4, 8) in which a somewhat similar colloidal gold curve was demonstrable. The tubes affected were always the first five, those of the so-called "syphilitic zone," sometimes six and seven, or more, tubes also reacted. This seems to indicate that the colloidal gold reaction in these cases was due to a syphilis toxin or some substance formed by its action, the blood serum already having reacted positively to the Wassermann test and no evidence of other provocative agents forthcoming to claim title to such a gold curve. In all the cases of positive Wassermann reaction in the spinal fluid, the Wassermann reaction was also positive in the blood serum. We have so far encountered no case of hereditary neurosyphilis in which the cerebrospinal

fluid showed a positive, and the blood serum a negative Wassermann reaction. Jeans [4] has reported one such case.

Along with these biochemical reactions, we have been able to show concomitant mental defect. While the psychologic reactions are those of the ordinary feebleminded or backward child, it is doubtful whether or not we can properly use the term mental defect in these cases, although one might be justified in some cases, especially those in which there is a pregenital or fetal aplasia, due to a potent "nascent form," possibly, of syphilotoxin. But in the milder degrees of syphilitic hypophrenia we are certainly not justified in using the term "defect," for with consistent and prolonged treatment, these cases improve, and the intelligence quotient rises correspondingly.

In the relatively older children, those born nearer the parental infection, the physical findings and Wassermann reactions on serum and fluid were not only positive, but they also showed a greater degree of mental deficiency. The children born later, showed less striking biochemical reaction, for although the majority were seropositive by the Wassermann test, their cerebrospinal fluid was usually negative and they showed less mental defect. The third class of children, the youngest born, those farthest removed from the original infection were, for the most part, entirely negative to laboratory tests and showed the least degree of feeblemindedness—but they were backward mentally to some extent.

We observe several psychic terraces ascending pari passu with the demonstrable grades of physical or chemical evidence, and it would seem that the milder forms of mental backwardness in the youngest and healthiest of these children, though syphilis is not demonstrable by laboratory test, is nevertheless due to the syphilis toxin, of too attenuated a form to demonstrate by test tube, but sufficient to register on a more sensitive indicator, the psyche. Furthermore if (as in Case 6) the first child, born before the parent's infection, is normal mentally and physically, and the father and mother are not feebleminded, it seems reasonable to exclude ordinary plasmic feebleminded-ness in the younger children born after, but too long after to cause a positive Wassermann. The deduction is that the same cause is operative here as in the older fraternity though the virus may be attenuated. Some careful observers [5] have felt that syphilis is merely incidental to and not genetic of the feeblemindedness.[6] The evidence furnished in family "B" (Table 3) runs counter to this view. Again, in

5. Kuhlmann, F.: Syphilis in Its Relation to Feeblemindedness, J. Lancet **38**:215, 1918.

6. Bevan, L.: Andriezen and Echeverria, quoted by Southard in Waverly Researches in Pathology of the F. M., Series 1, p. 43

the study of the pathology of the feebleminded[7] Southard, Fernald and Taft found suspicious evidence of syphilis in the brains of hypophrenics whose serum was negative to the Wassermann test, and in a larger number of cases than is generally suspected. Mott,[8] reporting twenty-two cases of juvenile general paralysis, says most of these patients were mentally deficient from birth.

An attempt has been made to show this graded attenuation of the virus as manifested by psychometric analysis (Fig. 1). All five children (Cases 1, 2, 3, 4, 5, Table 2) of one family are here represented with complete laboratory and psychometric analysis. The consistent rise of the intelligence quotient will be noted in the first three children.

TABLE 2.—SEROLOGY OF FAMILY A (CASES 1, 2, 3, 4, 5 IN TABLE 1)

	Age, Yrs.	Mental Age, Yrs.	Intelligence Quotient	Wassermann Reaction		Globulin	Albumin	Cells	Colloidal Gold Test	Remarks
				Serum	Cerebrospinal Fluid					
Father	..	....	..	+	+	2	2	269	5555443210	Paresis
Mother	..	....	..	+	..	...	..	...	.....	Has since developed paresis
Case 1	13	6 8/12	51	+	+	0	Normal	1	5555441000	
Case 2	12	8 8/12	72	—	—	0	Normal	0	0000000000	Feebleminded
Case 3	9	7 4/12	81	+	—	0	Normal	0	0012100000	Feebleminded
Case 4	8	4 10/12	60	+	—	0	Normal	0	0033310000	Feebleminded
Case 5	4	3 8/12	91	—	—	0	Normal	10	0000000000	Backward mentally

TABLE 3.—SEROLOGY OF FAMILY B (CASES 6, 7, 8, 9, TABLE 1)

	Age, Yrs.	Wassermann Reaction		Globulin	Albumin	Cells	Colloidal Gold Test	Remarks
		Serum	Cerebrospinal Fluid					
Father	..	+	—	0	Normal	0	0000000000	No symptoms
Mother	..	—	..	...	.....	...	.....	Apparently healthy
Case 6	16	—	..	...	.....	...	.....	Case 6 born in Italy, mentally and physically sound; father contracted lues after emigration
Case 7	12	+	+	0	Normal	37	5555531000	Case 7 born in America; observation case; manic state
Case 8	6	+	—	0	Normal	1	1111210000	Case 8, feebleminded
Case 9	5	—	—	0	Normal	0	0000000000	

The fourth child shows an exceptional variation, dropping below the third child. In the fifth, however, the intelligence quotient rise is again resumed and carried almost to normal.

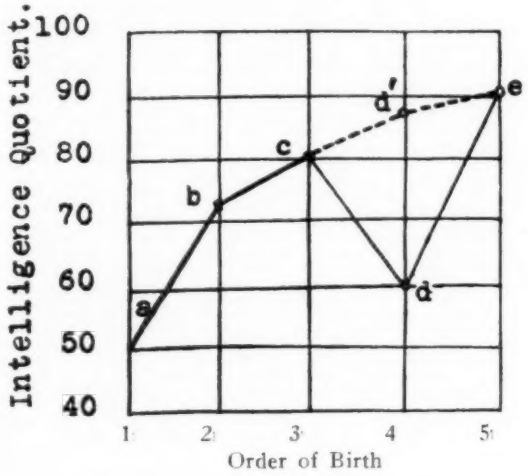
Just why there is a drop in the fourth case is not evident. Several factors may come into play here. A syphilitic meningitis affecting

7. Fernald, W. E.; Southard, E. E., and Taft, A.: Waverly Researches of the F. M. (Memoirs of Am. Acad. of Arts & Sc.) **4**:117 (May) 1918. (Exudative Lesion Frequent in Stable Institution Series.)

8. Mott, F. W.: Arch. Neurol. **1**:250.

more or less generally the cortical ganglion cells, or some other infectious meningitis, possibly, complicating childhood disease [9] might have augmented the mental defect. In the case of the second child one would have expected a positive Wassermann on the blood serum. This boy [10] was tested but once, whereas the other children's tests were all verified by retest.

Thus in the accompanying figure the curve representing the relative degree of syphilogenic injury to successive children as reflected by psychometric tests in a family in which there is parental syphilis (other adverse influences may react similarly) is shown by the letters a, b, c, d, e, in which the vertical scale represents the intelligence quotient and the base line represents the order of birth. The fourth child drops below in his mental tests and he also (Table 2) shows a



more marked colloidal gold reaction (tests were done the same day with the same reagents). We believe therefore, that this child is an exception, and that otherwise he would have graded somewhere between 80 and 90. The curve would then read as shown by the letters a, b, c, *d'*, e. On the physical side, the progressively decreasing strength of the syphilitic virus is represented by a parallel curve more roughly shown by the blood and spinal fluid analyses;[11] so that this graded attenuation is elicited both pathologically and clinically, the psychical phenomena running abreast the physical reactions in nice correlation.

In the next family (Table 3, Cases 6, 7, 8 and 9), a regular degradation of the syphilotoxin in succeeding members of the fraternity after the infection in the parent is again shown by actual test.

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9. Skoog, A. L.: Brain Complications in Measles, J. A. M. A. **74**:1697 (June 19) 1920.

10. Committed to a distant truant school, he was not easily available.

11. See Table 2; also the same curve in Family B, Table 3.

The oldest child was born before the father emigrated to America, where he contracted syphilis. The next child (Case 7) was the first born after the mother and older daughter joined him several years later. This was a girl, in good physical condition, who was brought to the hospital for observation in a manic state. The laboratory tests as shown were all positive for neurosyphilis. The next child in order of birth (Case 8) showed a positive Wassermann reaction in the blood, a negative Wassermann reaction in the fluid, but a trace of suspicious evidence in the syphilitic zone of the colloidal gold test. The next child in order (Case 9) gave entirely negative reactions, both on the blood serum and spinal fluid. The physical attenuation curve is here regularly drawn both as to Wassermann reaction and the colloidal gold test.

The correlations of mental age with physical tests are unsatisfactory or impossible here because psychometric figures are incomplete (Case 9) and the unreliability of the mental age in Case 7 (manic state with rapid-fire associations).

SUMMARY

Blood and spinal fluid examinations have been made on twenty-two children, born of syphilitic parentage. It has been found that various degrees of syphilitic infection are present in a definite order in a family of several children, the oldest, born soonest after the parental infection, showing the greatest injury and the succeeding children showing milder degrees in order.

Psychometric examinations done on these children show corresponding psychic terracing going pari passu with the grades of physical defect.

The youngest children of a family though seronegative, have been found to be feebleminded to a slight degree. The syphilitic injury to the nervous system either projects beyond the physical defect or by reason of the peculiar reactions of the brain compared with other organs we have a finer indicator in it than in ordinary tissues. Or, again, while the central nervous system may recover more rapidly in succeeding births as registered by the Wassermann, colloidal gold and other tests, the recovery may not be as complete.

CONCLUSIONS

Mental deficiency in congenitally syphilitic children of not feebleminded parents is in the majority of cases due to syphilis.

The virulence of the congenital syphilis toxin in a given fraternity as evidenced by psychometric analysis and biochemical test varies inversely with the order of birth.

THE ELECTROCARDIOGRAM IN NORMAL CHILDREN*

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Electrocardiography is that phase of cardiography which deals with the direction, the time relations and the magnitude of the "action currents" of the heart.[1] It does not register the strength of the contraction of the heart muscle, but it is merely the expression of the electrochemical processes concerned with the contraction of the heart muscle.

Electrocardiography in children is a large and fertile field for clinical research. In spite of the many and noteworthy contributions that have been made to the diseases of circulation in general with the aid of the electrocardiograph, no attempt has as yet been made in this country at a systematized and comprehensive investigation of this subject in children. In the electrocardiograph there are great possibilities for a study of physiologic peculiarities of the normal heart of the child. Through it we can obtain a clearer conception of the peculiarities of the heart mechanism during the different periods of growth. The preciseness of this method will allow us to correlate more accurately certain clinical symptoms with their associated arrhythmias and other forms of impulse disturbances. Since the electrocardiogram is an expression of the muscle rather than of the valves of the heart, we may obtain data, otherwise unobtainable, about the heart muscle, and we may hope to further our knowledge of the prognosis of heart disease, about which we know practically nothing.

This paper is the first of a series on this subject. It includes (1) the study of the form of the curves; (2) the heights of all deflections; (3) the rate; (4) the rhythm; (5) the transmission time, and (6) the relation between the right and left ventricles of the heart during the period of childhood. Electrocardiograms of 101 children (including prematures), from one hour to 13 years of age, are recorded. They were all normal as far as clinical examinations could determine.

The first investigation of the electrocardiogram in the child was undertaken in 1908 by Funaro under the direction of Nicolai.[2] He

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1. Lewis, T.: Electrocardiography and Its Importance in the Clinical examination of Heart Affections, Brit. M. J. **1**:45, 1912; **2**:1421; 1479, 1912.

2. Funaro and Nicolai: Electrocardiogram of Nurslings, Zentralbl. f. Physiol. **58**: 1908.

studied forty-five children, most of whom were infants. He was the first one to describe the deep S in Lead I in nurslings. This has since been corroborated by other observers. His work is open to criticism in that he employed only the first lead, and he does not state whether his subjects are normal or abnormal.

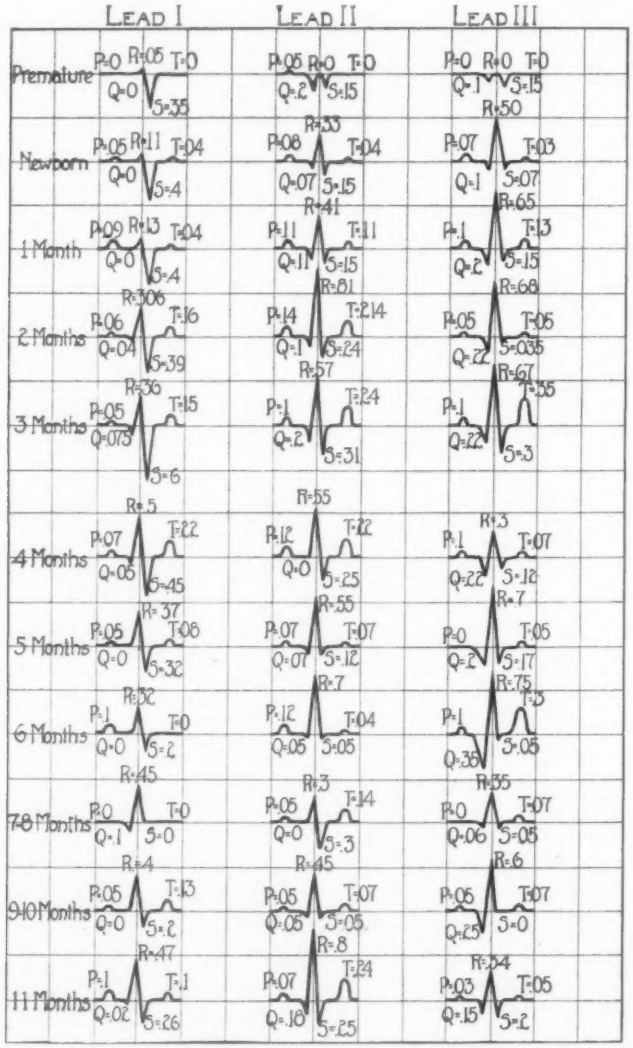

Fig. 1 a

Fig. 1. — Composite electrocardiograms of normal children of all ages. Exact heights of all deflections in millivolts. Each square represents 5 scale divisions of 0.5 mvlt.

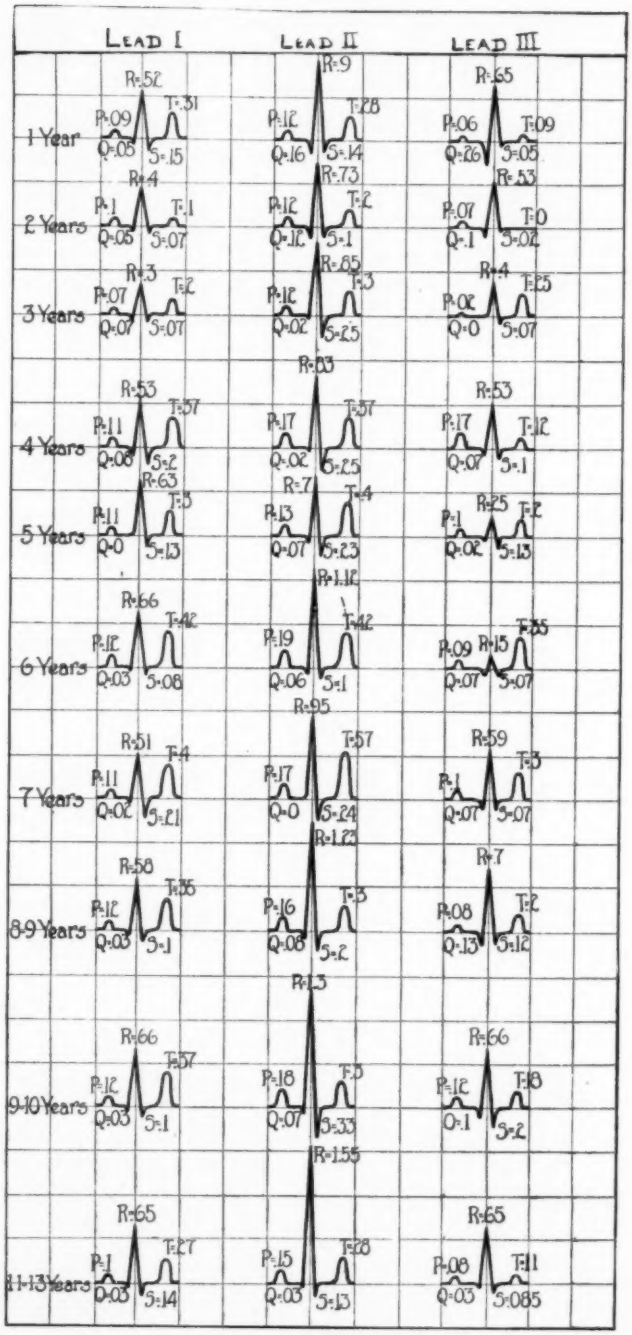
LEAD I
LEAD II
LEAD III
1 Year
2 Years
3 Years
4 Years
5 Years
6 Years
7 Years
8-9 Years
9-10 Years
11-13 Years

Fig. 1 b

A little later in the same year, Heubner [3] made a similar study. He also took tracings of the first derivation only. He either waited for his subjects to fall asleep or he administered chloral hydrate to keep them quiet. He also noted the deep S and the very small or absent R in new-borns. He thought that the change from the infantile type of electrocardiogram to that of the adult occurred at about the eleventh or twelfth month. Hecht,[4] in 1912, contributed the first comprehensive treatise on this entire subject. Although in this work he made a complete study of the physiologic peculiarities of the heart of the child, he did not establish a composite curve that could be used as a guide for the normal at all ages of childhood.

Krumbhaar and Jenks,[5] in 1916, made a study of forty-five normal children of all ages, from which they have derived their composite electrocardiograms. With the exception of the work of Hecht (1912) and Krumbhaar (1916), there has been no attempt to investigate the peculiarities of each of the deflections of the normal electrocardiogram of the different periods of childhood.

TECHNIC AND PROCEDURE

It is much more difficult to employ the electrocardiograph in children than in adults. For this reason it is especially important to mention some of the obstacles one meets in working with children. The subject is connected by means of copper electrodes to the switchboard. The right arm, left arm and left leg are used. I used for the new-born small, tightly fitting copper cuffs held together by rubber bands. For the older children the electrodes used for the adult are suitable.

Absolute quiet is the most important factor in securing accurate tracings. In the new-born it is better to take the tracings while the baby is asleep. A nipple in the mouth for a few minutes before the experiment will keep the baby quiet long enough to take a satisfactory record. In working with infants, it is very hard to keep the string out of the field, and it is especially important to keep the string neither too tight nor too loose. The slightest movement due to coughing, sneezing or crying will cause marked deflection of the string. Such a tracing must be discarded. In young nurslings the attention can be held by a lighted match or a bright object of some kind.

It is also important to wash carefully with soap and water the

3. Heubner: Elektrokardiography des Säuglings und des Kindes, Monatschr. f. Kinderh. **7**:66, 1908.

4. Hecht, A.: Der Mechanisms der Herzaktion im Kindesalter, Ergebn. d. inn. Med. u. Kinderh. **11**:324, 1913.

5. Krumbhar, E. B.: Electrocardiographic Studies in Normal Infants, Heart, **6**: 1915.

parts which come in contact with the electrodes, for the oil in the skin increases the resistance to the current. There seems to be about as much skin current generated by children as by adults, and this must be exactly compensated by throwing an equivalent amount of current into the circuit in the opposite direction.

The position of the electrodes must be watched carefully, especially in nurslings. The slightest wriggling will loosen the electrodes and cause either low deflections or none at all. They must be held firmly, preferably by means of rubber bands. The bare legs or arms must be kept from touching each other so that there will be no crossing of the current.

All of the records were obtained either at the heart station at Millard Hall at the University of Minnesota or at the University Hospital 400 feet away from the electrocardiograph. When the work was first undertaken, at least two tracings were taken at each sitting, and in some instances three or even more. It was found that although the height of the deflections differed slightly in the same subject, the form was quite constant. It was not necessary to administer any drugs (as was done by some of the above mentioned investigators) in order to obtain satisfactory records. All three leads were taken for each child. Only those who were within the normal limits of weight and height, with normal shaped chests, and hearts that showed no abnormal signs were selected. In this study the same technic was used with all children. The child lay on the back, the room was perfectly quiet, and the time was usually between 9 and 11 a. m., after the first feeding. The same electrodes were always used. The pads for the electrodes were boiled after being washed in soap and water, and before being applied were immersed in saline solution. The string was so adjusted that one millivolt of current caused a deflection of one centimeter. Platinum strings were used of about 3,500 ohms resistance and 0.02 second deflection time. The accompanying tables are calculated on the basis of one millivolt—one centimeter. The body resistance does not vary appreciably in new-borns and older children.

P Wave.—It is the consensus of opinion among all authorities that the P wave represents auricular activity. Although there are exceptions to the rule, the height of P may be considered as representative of the summation of action currents in the auricles, while the distance from the beginning of P to the beginning of R (the so-called P-R interval) is representative of the period of impulse conduction through the auricles and through the conducting tissues to the ventricles.

Hecht [4] has attempted to establish a normal P for childhood, by dividing the height of R by the height of P of the same lead, thereby

getting what he calls the auricular quotient. He found that in new-borns the quotient was 68 per cent., and in older children (taking the average of all children aged from 2 to 13 years) 12.5 per cent. I do not believe that this can be used as a reliable guide for the height of the P. In the first place, he used only derivation I, whereas throughout childhood P in Leads II and III is higher than in Lead I; secondly, in order to have an accurate guide for all ages, it would be necessary to divide childhood into four periods at least, instead of two, as is done by Hecht; thirdly, in very young infants, especially in the new-born, the R in Lead I is small and that would decrease the quotient for the entire four periods of childhood. Linetzky, in working with adults only, found that P did not vary with age. His averages for the height of P from 20 to 50 years were from 1.2 to 1.3 mm.

In my investigations I found the following: (Fig. 2).

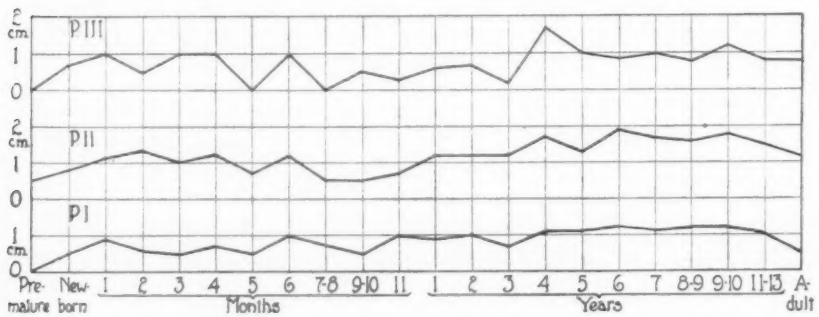

Fig. 2.—Height of P deflection. Each square represents 0.5 mvlt. or 5 scale divisions.

In the two prematures none of the three leads showed the auricular deflection. From birth through the entire period of childhood P is constant and definite. In only two of the entire series was P absent. In general, the P is higher in childhood than in the adult. In derivation one, the average height during the first two years is 0.6 mm. (+) and from 2 to 13 years it is 1.0 mm., whereas in the adult it is 0.5 mm. In this lead during the first few months of life, P is higher than in the adult, although it is not quite so broad. In Lead II, the average for the period of infancy is 1.2 mm. and for the latter part of childhood it is 1.5 mm. In this derivation, the P is also higher throughout childhood than in later life. Beginning with a height of 0.5 of a scale division [6] at birth, it abruptly increases during the first few months to slightly over one scale division. During the rest of childhood it remains between 1 and 2 mm. as compared to an average of 1.2 mm. in the adult. The height of this deflection in this lead is

6. (+) 1 mm. = 0.1 m volt = 1 scale division.

less constant than in the other two leads. Of the entire series, I found either a diphasis or an inverted P wave in eight records; six of these were in new-borns, one in a child 3½ years old, the other in that of one six years old. In two cases P_1 and P_3 were inverted. Four of these latter were diphasic and one was very broad, being almost flat at the top.

The clinical significance of an electronegative P has not been definitely established. Carter in an investigatiion of the inverted P in derivation three concluded that it was caused by (1) an ectopic rhythm as the result of a definite change in the location of the pacemaker, and by (2) disturbances in the respiratory rhythm. He found that, especially in Lead III, P was very much influenced by the different phases of respiration; that the P was lower at the end of respiration than at the beginning; and that the slight rotation of the heart about the sagittal axis of the body, which may be brought about by a very deep

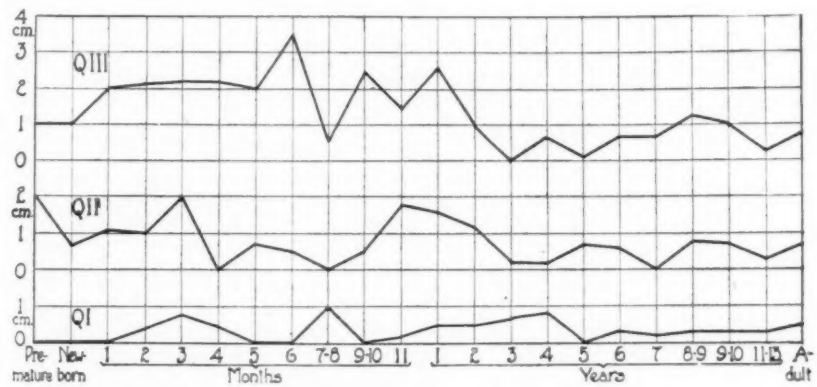

Fig. 3.—Height of Q deflection. Each square represents 0.5 mvlt. or 5 scale divisions.

inspiration causing a change in the position of the heart, also changed the height and shape of the P.

Goddard[7] noted the great frequency of an inverted P_3. In 700 cases P_1 was inverted in four, P_2 in seven, and P_3 in seventy-five.

DISCUSSION

If we can assume that there is any direct relation between the size of the P and the structure of the auricle, then we can readily explain the relatively high P during the first year of life by our anatomic data. We know that the foramen ovale in a large number of instances continues to remain open until the end of the first year. This may

7. Goddard: Changes in the P wave of the Human Electrocardiogram. Arch. Int. Med. **16**:632 (Oct.) 1915.

increase the work of the auricles during that time. Gundobin [8] states that the auricles during the first year are from 21 to 22 per cent. of the weight of the ventricles as compared to 20, 18 and 17 per cent. in the following years.

There is no greater frequency of abnormal P-waves in this series than in those reported on normal adult hearts.

Q Wave.—There has been and still is a difference of opinion as to the physiologic significance of the Q deflection. Q is a downward deflection at the beginning of the ventricular complex. It is not always present. It is important in the electrocardiogram of childhood. Hoffmann,[9] who seems to have the most commonly accepted explanation for the Q deflection, believes that it represents the beginning of the ventricular impulse conduction. According to Lewis,[10] the Q in Lead I represents the spread of left ventricular activity, and in Leads II and III he believes it to be a right ventricular event.

Krumbhaar [5] noted the relatively deep Q in childhood, especially in Leads II and III. The average of the Q in these leads is less than 1 mm. in the adult; whereas in the infants he examined he found the Q relatively and absolutely deeper.

In my series I found also, that, in general, the Q of Leads II and III was deeper than in the adult (Fig. 3). In Lead I, up to the first month, Q is absent; from the first to the fifth month, the Q is variable and indefinite, never reaching deeper than 0.7 mm. From the eleventh month on, there is a gradual increase of Q which averages about the same as in the adult. From the fifth year there is a remarkable constancy which persists throughout the rest of childhood, averaging about 0.4 of a scale division. This fact, namely, that during the first year the Q_1 is relatively of diminished depth and in the majority of instances is absent, and that after the first year and during the rest of childhood Q_2 and Q_3 become larger, the average being equal to that of the adult, corroborates the theory of Lewis that Q_1 represents left ventricular activity. Q_2 is not quite so uniform in its depth. During the first three months it is rather deep, running on an average of one scale division. From the fourth to the ninth month, it remains more uniformly at the level of one-half of a scale division, and from the ninth month to the second year it increases to two mms. From the third year on, it averages 0.5 mm. which is slightly less than in the adult. Q_3 is deeper during the first period of life than Q in

8. Gundobin: Die Besonderheiten des Kindesalters, Berlin, 1912.

9. Hoffmann, A.: Die Arythmie des Herzens im Elektrokardiogramm, München. med. Wchnschr. **44**:1909; Herz und Konstitution, Jahreskurse fuer aertzl. Fortschr., February, 1918.

10. Lewis, T.: Observations on Ventricular Hypertrophy, Heart **5**:367, 1914.

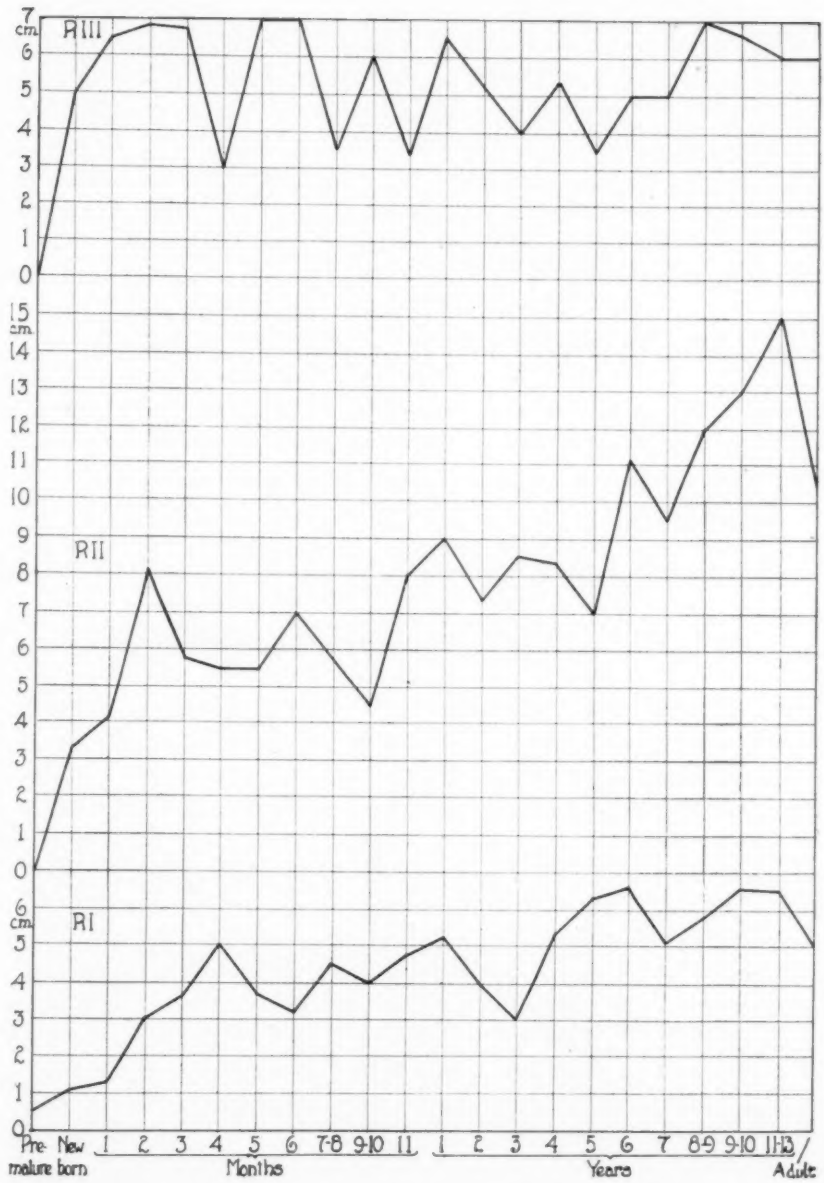

Fig. 4.—Height of R deflection. Each square represents 0.5 mvlt. or 5 scale divisions.

the other two leads. From the fifth month to the second year, it is somewhat variable. At the second year, there is a sudden diminution and the figure remains low throughout the rest of childhood. The average for the first two years is 0.9 of a scale division. From the age of 2 on it is 0.6.

DISCUSSION

If the theory of Lewis is correct, that Q_1 represents left ventricular activity and Q_3 right ventricular activity, then the depths of this deflection in the third derivation, as shown in Figure 3, give corroborating evidence of right ventricular preponderance in nurslings.

R Wave.—The R wave is the most constant and important electropositive deflection of the ventricular complex. There is still, however, some difference of opinion as to the physiologic significance of this wave. Hoffman [11] is of the opinion that the R wave represents the impulse conduction through the ventricular fibers or the excitation of the muscle which immediately precedes the actual contraction.

Linetsky [12] found that in adults the R becomes higher with age and with the increase of the heart size. He found the average height in a series of 300 adults to be from 11 to 14 mm. (Lead II). In my study, I found that in childhood (Fig. 4) the R generally becomes much higher than it is in the adult, although in Lead I the R is lower in the premature and in the new-born than in any other period of life. From the first month there is an abrupt ascent which reaches its peak in the fourth month. From then on, the averages form an up and down curve until the fourth year. During the second and third year, I have used the averages of only two cases. This may account for the unexpected drop of the R wave after there had been an ascent (Fig. 4). After the fourth year, the high R is suggestive of a change to a left ventricular preponderance.

R in Lead II shows a very definite ascent which is consistent with the anatomical change in the mass relationship between the right and left ventricle. No R_2 was found in the premature, but immediately after birth the R_2 rises very rapidly. From the first to the fourth year the curve is about the same. From the fourth year on, there is another increase which passes the adult height at six years. From then to the end of childhood it becomes higher and higher, so that at puberty it averages 15 mm. as compared to 10.3 mm. in the adult.

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11. Hoffmann, A.: Demonstrationen zur Lehre von der Form des Elektrokardiogramms, Verhandl. d. deutsch. Kong. f. innere Med. **18**:429, 1911.

12. Linetzky. Die Beziehungen der Form des Elektrokardiogramms zu dem Lebensalter, der Herzgroesse und dem Bludrucke, Ztschr. f. exper. Path. u. Therap. **9**:669, 1911.

R_3 is variable throughout the entire period of childhood. It is absent in the premature, but during the first month of life it reaches a rather high measurement which it retains until the fourth month. For some unknown reason it falls and stays at a low level until the eighth month. From then on, until the fourth year, it is again variable. After that it increases and remains high throughout the rest of childhood. For the first two years it averages 5.5 mm.; from then until 13 years it averages 5 mm.

S Wave.—The S is still a subject of dispute. It is not always found in the normal electrocardiogram. In Lead I, S is a right ventricular event, and in Lead III it is a left ventricular event. So far

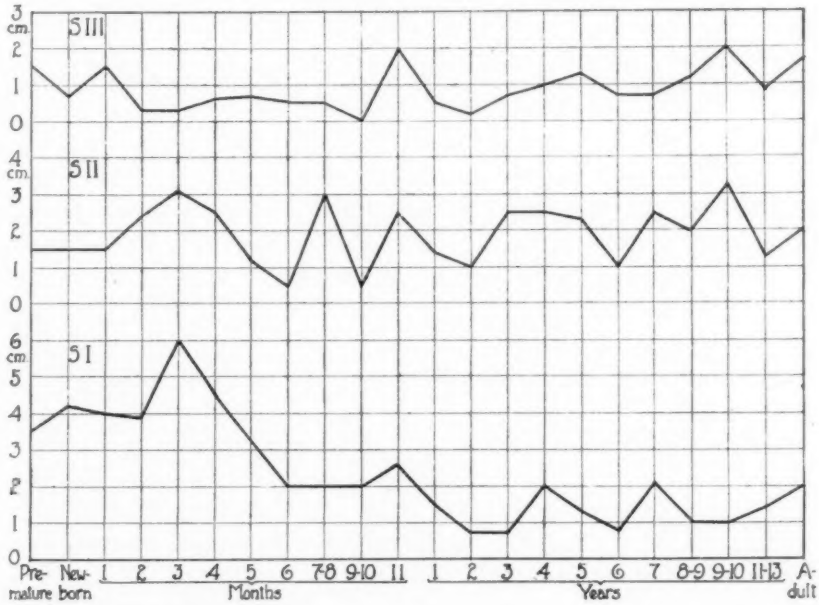

Fig. 5.—Height of S deflection. Each square represents 0.5 mlvt. or 5 scale divisions.

back as 1908 Funaro,[2] working with Nicolai, found a deep S in a large majority of the forty-five infants examined. Nicolai concluded that this was due either to a left ventricular hypertrophy or to a more horizontal position of the heart at this age.

At almost the same time Heubner[3] also found this same characteristic deep S_1. There is no difference of opinion regarding the fact that S_1 is deep during the first few months of life. There is some difference, however, as to when the S disappears. Some believe it disappears as early as the third month, others at about the seventh or eighth month; others, again, are not sure of a permanent change

until the end of the first year. There are also differences of opinion among the earlier observers as to the explanation for this characteristic curve. Is it due to the higher and more horizontal position of the heart on account of the high diaphragm? Or, is it due to the fact that in this period the right ventricle is relatively preponderant over the left? I have been able to confirm the opinion of Grau [13] and others that change of position does not have any influence on the shape of the S wave. On the other hand, using the formula of White [14] $(U_1 + D_3) - (D_1 + U_3) = \text{constant}$, to determine the ventricular preponderance and comparing the electrocardiograms of the period of infancy month by month with the size and thickness of the ventricles of the same ages, one is justified in concluding that in the anatomic relationship between the right and left ventricles lies the explanation of the deep S in Lead I.

In my series, S_1 is both relatively and absolutely greatest during the first three months of life (Fig. 5). From its peak at the end of the third month it diminishes abruptly to the sixth month. Then holding a level until the tenth month, it has another diminution until it reaches its minimum deflection at about the beginning of the second year. From then on it increases slightly and remains about the same throughout the rest of childhood. In the latter part of childhood it is slightly deeper than in the adult.

Lead II is generally lower during the period of infancy than in later childhood, although the differences between the periods is much less than in Lead I. The average from three years on is about one scale division higher than during the period of infancy.

Lead III shows a curve somewhat similar in its inconstancy to Lead II. The average of S_3 for the entire childhood is lower than that of S_2. From birth to the third year the curve is too variable to draw conclusions from; but from the third year on, the curve is more constant and averages less than that of the adult.

The influence of crying, respiration and position on the shape and size of S have all been studied. The occurrence of a deep S_1 in older children will be discussed in another paper in connection with atypical electrocardiograms in normal children.

T Wave.—The T wave is the last of the ventricular complex. There is still a difference of opinion as to its meaning.

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13. Grau, H.: Ueber den Einfluss der Herzlage auf die Form des Elektrokardiogramms, Ztschr. f. klin. Med. **69**:281, 1909; Zentralbl. f. Physiol. **23**:440, 1910.

14. White, P., and Block, A. V.: Electrocardiographic Evidence of Abnormal Ventricular Preponderance and of Auricular Hypertrophy, Am. J. M. Sc. **156**: 17, 1918.

Linetzsky believes that in the adult the T wave decreases with the increase of age, especially in the very old. He found that at about 45 years of age it was quite constant, averaging 3 mm. From then on, it decreased to 2.1 mm. The decrease of the S was in direct proportion to the increase of the R wave. Krumbhaar in his work with children found no T in the first week of life, but he found it constantly after the third week highest in Lead II and lowest and frequently inverted in Lead III. Hecht found it present, although not uniformly, in the new-born.

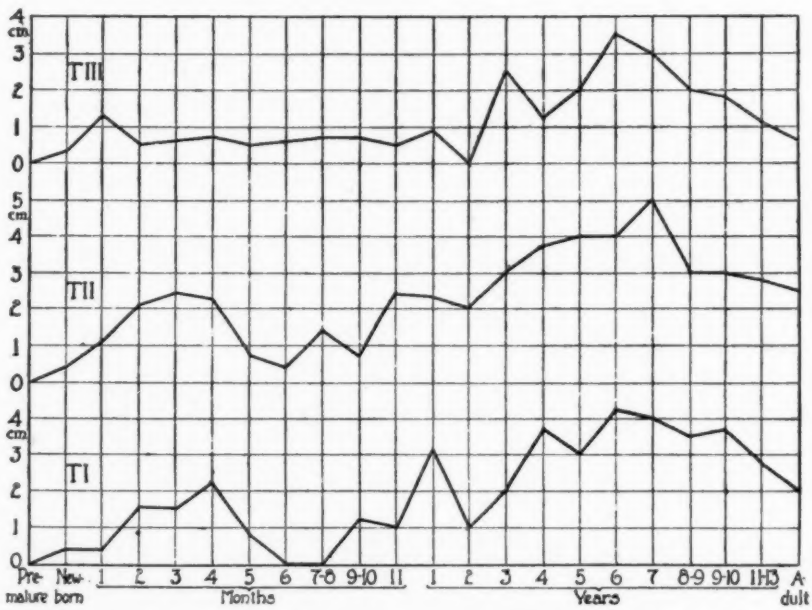

Fig. 6.—Height of T deflection. Each square represents 0.5 mlvt. or 5 scale divisions.

In the child the T is the phase of the ventricular complex most susceptible to unexplained changes. In my series of twenty-two cases I found no T in the premature, but right after birth, for the first ten days, it was present in 39 per cent. of the cases in Lead 1, 77 per cent. in Lead II, and 47 per cent. in Lead III (Fig. 6). After the first month it was quite constant. T_1 is more regular in its developmental changes than in the other two leads.

In Lead I, from the first to the fourth month, there is a sudden increase; then a sudden drop to the seventh month, and again an increase to the second year. I can see no physiologic explanation for such a change. From the second year on there is again an increase which after the fourth year remains about the same. In this lead

TABLE 1.—Twenty-Two Normal Electrocardiograms of Infants Aged from 1 Hour to 11 Days

No.	Age	Weight, Gm.	Sex	P			Q			R			S			T			Rate
				1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	
1	Premature 7 mo. 3 da.	2,110	M	0	0.1	0	0	0.3	0.2	0.1	0	0	0.4	0.2	0.1	0	0	0	176
2	Premature 8 mo. 1 wk	2,310	M	0	0	0	0	0.1	0	0	0	0	0.3	0.1	0.2	0	0	0	180
3	4 hours	3,115	M	0	0	0	?	0.02	0	0.3	0.32	0.15	0.3	0	0	?	0	0	110
4	8 hours	3,440	M	0.09	0.1	0.1	0	0.15	0.25	0.15	0.5	0.8	0.5	0.15	0.09	0	0	0	108
5	16 hours	2,840	M	0.1	0.15	0.05	0	0	0.1	0	0.3	0.45	0.25	0.2	0.2	0.18	0.02	0	152
6	18 hours	2,440	M	0.10	0.05	0	0	0.1	0.05	0.05	0.5	0.3	0.3	0.1	0	0	0	0	160
7	24 hours	3,600	M	0.05	0.15	0.05	0	0.01	0	0	0.22	0.35	0.22	0.15	0	0	0	0	102
8	2 days	3,230	F	0.1	0	0	0	0.05	0	0.2	0.3	0.75	0.3	0.2	0	0	0	0.05	135
9	2 days	3,125	M	0	0	0	0	0.1	0.1	0.1	0.25	0.3	0.3	0.15	0.05	0	0	0.09	
10	2 days	2,940	F	0.05	0	0.05	0	0	0	?	0.05	0.09	0.3	0	0.09	0	0	0	128
11	2 days	3,625	F	0	0.1	0.08	0	0	0	0.1	0.1	0.09	0.4	0.09	0.1	0	0.1	0	128
12	3 days	3,095	M	0.05	0.1	0.1	0	0.1	0.1	0.1	0.4	0.4	0.6	0.15	0	0.05	0.05	0.1*	
13	3 days	3,250	F	0.1	?	0.1	*	?	0.2	0.3	*	0.9	0.7	?	0.2	0.2	?	?	
14	4 days	3,810	M	0.1	?	0.1	0	?	0	0.2	*	1.0	0.7	*	0	0	0	0	132
15	4 days	3,100	M	0.1	0.15	0.1	0	0.1	0.1	0.1	0.4	0.5	0.5	0.2	0.05	0.1	0.1	0.09	130
16	4 days	2,800	M	0.15	0.1	0	0	0.1	0	0.5	0.3	0.2	0.4	0.3	0.2	0.05	0	0	140
17	5 days	2,790	F	0	0.05	0	0	0.05	0.05	0.05	0.1	0.1	0.3	0.1	0	0	0	0	174
18	5 days	3,420	M	0.1	?	0.1	0	?	0.2	0.3	?	0.9	0.7	?	0.2	0.2	?	0	135
19	5 days	2,720	M	0.05	0.1	0.08	0	0.05	0.15	0.1	0.2	0.3	0.5	0.05	0.05	0.05	0.09	0.08	202
20	6 days	3,440	F	0	?	?	0	?	?	0	?	?	0.2	?	?	0	?	?	172
21	9 days	4,000	F	0	0.15	0.05	0	0.1	0.1	0.1	0.5	0.4	0.3	0.3	0.2	0	0.1	0.09	160
22	9 days	4,000	F	0	0.2	0.25	0	0.15	0.3	0	0.5	0.7	0.5	0.3	0	0	0.1	0.5	180

One scale division represents 1 mm. in actual height and 0.1 mvlt. in potential.
* indicates inverted.

TABLE 2.—Forty-Five Normal Electrocardiograms of Infants Aged from 11 Days to 1 Year

No.	Age	Weight, Gm.	Sex	P			Q			R			S			T			Rate
				1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	
1	11 days	2,815	F	0	0.05	0.1	0	0.05	0.1	0.05	0.04	0.5	0.4	0	0.05	0	0.1	0.2	148
2	13 days	3,200	F	0.1	0.09	0.1	0	0.1	0.2	0.1	0.5	0.6	0.5	0.2	0.3	0	0.1	0.1	136
3	18 days	2,440	M	0.1	0.05	0	0	0.1	0.05	0.05	0.5	0.3	0.3	0.1	0	0	0	0	160
4	24 days	3,360	M	0.1	0	0	0	0	0	0.15	0.15	0.2	0.6	0.15	0	0	0.05	0.09	132
5	3 wks.	3,800	M	0.1	0.15	0.1	0	0.1	0.2	0.3	0.8	0.6	0.4	0.3	0.1	0.1	0.2	0.1	124
6	3 wks.	3,600	M	0.1	0.1	0.1	0	0.2	0.3	0.1	0.4	0.9	0.3	0.1	0.2	0.05	0.1	0.1*	128
7	5 wks.	4,115	F	0.05	0.1	0	0.15	0.3	0.1	0.3	0.9	1.2	0.7	0.5	0.6	1.5	0.4	0.3	136
8	5 wks.	4,030	M	0.15	0.2	0.1	0	0	0	0.1	0.6	0.8	0.4	0.3	0.1	0.1	0.1	0.1	128
9	6 wks.	3,840	F	0.1	0.15	0.1	0	0.3	0.4	0.5	1.0	0.9	0.6	0.4	0.1	0.2	0.2	0.2*	116
10	6 wks.	3,900	F	0	0.1	0.05	0	0.25	0.3	0.3	1.0	1.0	0.4	0.3	0.15	0.05	0.05	0	147
11	6 wks.	3,143	F	0.1	0.15	0.05	0	0.1	0.15	0.15	0.6	0.8	0.6	0.1	0	0.15	0.2	0.1	165
12	6 wks.	3,820	F	0.1	0.15	0.09	0	0.05	0.1	0.35	0.4	0.4	0.4	0.25	0	0.15	0.1	0.1	140
13	6 wks.	3,720	F	0.05	0.1	?	0.05	0	?	0.3	0.3	?	0.1	0.1	?	0.05	0.05	?	
14	6 wks.	3,915	F	0.05	0.05	?	0	0.15	?	0.15	0.6	?	0.35	0.45	?	0.1	0.15	?	
15	2 mos.	4,300	M	0.1	0.1	0	0.05	0.1	0.2	0.3	0.8	0.6	0.3	0.1	0.1	0.1	0.4	0	160
16	2 mos.	4,500	M	0.1	0.09	0	0.1	0.2	0.4	0.2	0.1	1.0	0.4	0.2	0	0	0	0	
17	2 mos.	4,560	M	0.1	0.1	0.1	0	0	0.2	0.5	0.55	0.4	0.8	0.25	0.3	0.1	0.2	0	180
18	7 wks.	4,320	M	0.05	0.2	0.05	0	0.05	0.15	0.5	0.3	0.3	0.4	0.25	0.05	0.2	0.4	0.1	150
19	7 wks.	4,020	M	0.05	0	0	0	0.1	0.1	0.2	0.25	0.1	0.35	0.2	0.05	0.1	0.1	0.1	
20	10 wks.	4,940	F	0	0	0.1	0.1	0.3	0.4	0.4	0.6	0.3	0.5	0.8	0.3	1.5	0.2	0.2	148
21	12 wks.	6,110	M	0.1	0.2	0.1	0.2	0.3	0.3	0.3	0.6	0.7	0.5	0.3	0.8	0.3	0.4	0.6	136
22	3 mos.	5,840	F	0	0.2	0.2	0	0.2	0.2	0.6	0	1.5	0.8	0	0.1	0	0.3	0.5	120
23	3½ mos.	6,120	M	0.1	0.05	0.09	0	0	0.15	0.3	0.3	0.2	0.4	0.2	0.15	0.15	0.05	0.05	117
24	4 mos.	6,580	M	0.1	0.2	0.15	0	0	0.3	0.6	0.8	0.4	0.5	0.3	0.1	0.3	0.4	0.1	150
25	4 mos.	6,850	M	0.05	0.05	0.05	0.1	0	0.15	0.4	0.3	0.2	0.4	0.2	0.15	0.15	0.05	0.05	130
26	5 mos.	7,100 Lbs.	M	0.09	0.1	0.1	0	0.05	0.05	0.4	0.4	0.2	0.4	0.1	0.5	0.15	0.1	0.1	150
27	5 mos.	15	M	0.05	0.05	0	0	0.1	0.09	0.5	0.5	0.2	0.4	0	0	0.1	0	0.12	135
28	5½ mos.	14	M	0.1	0.05	0	0	0.1	0.25	0.3	0.65	0.7	0.25	0.2	0.15	0	0	0	125
29	6 mos.	14	M	0.1	0.2	0.1	0	0.1	0.35	0.45	1.15	1.1	0.2	0	0	?	0.4	0.3	135
30	6 mos.	5,320 Gm.	M	0.1	0.05	0	0	0	0	0.2	0.3	0.4	0.2	0.1	0.1	0	0.05	0.1	
31	8 mos.	15	F	0	0.05	0	0.1	0	0.08	0.4	0.3	0.3	0	0.3	0.1	0	0.2	0.1	135
32	8 mos.	18	F	0	0.05	0.01	0.1	0	0.05	0.5	0.3	0.4	0	0.3	0	0	0.15	0.05	
33	10 mos.	20	F	0.05	0.05	0.05	0	0	0.1	0.2	0.5	0.3	0.1	0.05	0	0.05	0.1	0.05	
34	11 mos.	19	F	0.1	0.1	0.05	0	0	0.05	0.6	0.7	0.3	0.15	0.3	0.2	0.2	0.3	0.05	110
35	11 mos.	17	F	0.09	0.1	0.09	0	0.1	0.15	0.3	0.6	0.7	0.35	0.3	0.25	0.15	0.1	0.09	180
36	11 mos.	17	F	0.1	0	0	0.1	0.1	0	0.55	0.8	0.25	0.2	0.3	0.2	0.2	0.25	0.1	110.5
37	11 mos.	19	F	0.15	0.2	0	0.1	0.25	0.1	0.4	0.1	0	0.2	0.3	0	0.15	0.25	0	120
38	13 mos.	21	F	0.05	0.1	0.15	0.1	0	0	0.8	0.7	0.4	0.2	0.25	0.1	0.2	0.25	0.2	135
39	14 mos.	18	M	0.1	0.2	0.05	0.15	0.4	0.5	1.0	1.2	1.0	0.1	0.1	0	0.6	0.5	0.2	140
40	14 mos.	22	M	0.1	0.1	0	0.1	0.3	0.4	1.1	1.7	1.0	0.1	0.35	0.15	0.3	0.2	0	105
41	15 mos.	22	M	0.1	0	0.05	0	0	0.2	0.4	0.4	0.8	0.2	0.05	0	0.2	0.1	0.1	105
42	16 mos.	24	M	0	0.1	0.1	0	0.2	0.2	0.35	0.9	0.7	0	0	0	0.1	0.15	0	135
43	19 mos.	27	M	0.1	0.1	0	0	0.1	0.2	0.45	0.5	0.4	0.2	0.2	0.1	0.2	0.15	0	145
44	19 mos.	29	F	0.2	0.2	0.1	0.1	0.2	0.2	1.0	0.7	0.6	0.2	0.2	0.2	0.3	0.3	0.2	110
45	1 year	20	M	0	0.1	0	0	0	0.1	0.5	1.0	0.55	0	0	0	0.2	0.25	0	115

TABLE 3.—Ten Normal Electrocardiograms of Children Aged from 2 Years to 5 Years Inclusive

No.	Age,	Weight, Lbs.	Sex	P			Q			R			S			T			Rate
				1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	
1	2 years	24	M	0.1	0.1	0.05	0	0.05	0.1	0.4	0.7	0.5	0	0	0	0.1	0.2	0.1	160
2	2 years	19	M	0.1	0.15	0.1	0.1	0.2	0.1	0.4	0.75	0.55	0.15	0.2	0.05	0.1	0.2	0	187
3	3½ years	31	M	0.05	0.2	0.05	0.1	0	0	0.3	0.5	0.2	0.1	0.3	0.1	0.2	0.4	0.1	88
4	3½ years	32	F	0.04	0.05*	0.05	0.05	0.05	0	0.3	1.2	0.6	0.05	0.2	0.05	0.2	0.5	0.1	82
5	4 years	35	F	0.15	0.05	0.1	0.15	0.05	0.15	0.6	0.7	0.6	0	0.15	0	0.5	0.4	0.15*	75
6	4 years	36	M	0.1	0.15	0.05	0	0	0	0.6	1.2	0.6	0.2	0.4	0.2	0.3	0.4	0.1*	83
7	4½ years	36	F	0.1	0.3	0.2	0.1	0	0.05	0.4	0.6	0.4	0.4	0.2	0.1	0.3	0.3	0.15	100
8	5 years	47	F	0.15	0.2	0.1	0.1	0	0	0.5	0.6	0.2	0	0.1	0.4	0.4	0.6	0.3	82
9	5 years	49	M	0.1	0.3	0.1	0	0.1	?	0.7	0.7	?	0.3	0.4	0.2	0.3	0.4	?	105
10	5 years	40	F	0.1	0.2	0.1	0	0.1	0.05	0.7	0.8	0.5	0.1	0.2	0.3	0.2	0.2	0.3	94

TABLE 4.—Twenty-Six Electrocardiograms of Children Aged from 6 Years to 13 Years

No.	Age, Yrs.	Weight, Lbs.	Sex	P			Q			R			S			T			Rate
				1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	
1	6	41	M	0.1	0.2	0.05	0	0	0	0.6	0.8	0	0.3	0.1	0.2	0.4	0.5	0.1*	97
2	6	47	M	0.15	0.25	0.15	0.1	0.1	0.05	0.7	1.1	0.4	0	0.1	0	0.4	0.4	0.2*	100
3	6	43	F	0.1	0.05	0.1*	0.05	0	0	0.6	0.8	0.2	0	0.2	0.15	0.4	0.3	0.3*	101
4	6	42	F	0.15	0.25	0.15	0	0.2	0.3	0.9	2.2	2.1	0.1	0.1	0	0.5	0.6	0.4	100
5	6	41	M	0.1	0.2	?	0	0	?	0.5	0.7	?	0	0	?	0.4	0.3	?	95
6	7	45	F	0.2	0.2	0.1	0.1	0	0.2	0.7	0.9	0.5	0.4	0.2	0	0.7	0.5	0.4	96
7	7	47	F	0.1	0.15	0.1	0	0	0.15	0.2	0.7	0.65	0.25	0.18	0.15	0.2	0.3	0.2	100
8	7	46	F	0.05	0.15	0.2	0	0	0	0.35	1.1	0.8	0.2	0.4	0.1	0.2	0.6	0.4	80
9	7	45	F	0.1	0.2	0.05*	0	0	0	0.8	1.1	0.4	0	0.2	0.5	0.5	0.9	0.2	101
10	8	49	F	0.1	0.08	0.1*	0	0	0.2	0.85	1.4	0.5	0.2	0.2	0.25	0.2	0.2	0.2*	80
11	8	49	F	0.1	0.2	0.15	0.1	0.1	0	0.5	1.3	0.9	0.1	0.3	0.2	0.3	0.4	0.2	96
12	8	50	M	0.15	0.2	0.1*	0	0.15	0.2	0.4	1.0	0.7	0	0.1	0	0.5	0.4	0.2*	96
13	9	58	M	0.1	0	0.1*	0.15	0	0	0.6	0.4	0.2	0.05	0.4	0.5	0.3	0.1	0.1*	82
14	10	61	M	0.1	0.15	0.15	0.1	0	0	0.5	1.2	0.9	0	0.2	0.1	0.3	0.6	0.15	80
15	10	65	M	0.1	0.3	0.1	0	0.1	0.1	0.5	1.2	0.6	0.1	0.4	0.3	0.3	0.9	0.1	88
16	10	68	M	0.15	0.1	0.1	0	0.1	0.2	1.0	1.2	0.5	0.2	0.4	0.2	0.5	0.6	0.3	100
17	11	64	M	0.1	0.1	0.1	0	0	0	0.4	0.8	0.4	0	0.05	0.1	0.3	0.4	0.1	90
18	11	68	M	0.15	0.1	0.1*	0.15	0	0	1.0	0.5	0.1	0.1	0.2	0.3	0.4	0.2	0.1*	80
19	11	74	M	0.1	0.15	0.1	0	0	0	0.5	0.95	0.8	0.2	0.2	0.1	0.2	0.2	0.2	113
20	11	70	M	0.05	0.2	0.1	0	0.2	0.2	0.35	1.4	0.9	0.2	0	0	0.1	0.3	0.1	110
21	11	71	F	0.1	0.1	0.05	0	0	0.02	0.6	0.75	0.4	0	0	0	0.1	0.2	0.05	82
22	12	78	M	0.1	0.15	0.1	0.1	0	0.1	0.8	1.2	0.6	0.2	0.5	0.2	0.4	0.5	0.1	108
23	13	84	M	0.1	0.2	0	0	0	0	0.9	1.6	0.7	0	0	0	0.4	0.4	0.1	100
24	13	72	M	0.05	0.2	0.1	0	0.05	0.1	0.45	1.2	1.3	0.2	0.2	0.1	0.3	0.4	0.15	108
25	13	96	M	0.2	0.2	0.10	0.1	0.05	0	1.6	1.6	0.9	0.3	0.2	0.1	0.4	0.3	0.15	95
26	13	84	M	0.1	0.1	0.05	0	0	0	0.4	0.4	0.2	0.2	0.3	0.1	0.4	0.1	0.1*	78

the average for the first two years is 0.9 mm. and the average from the second to the thirteenth year is 3.4 mm. The average for the adult, according to Lewis, is 2 mm. In Lead II the T is, on the whole, higher than in Lead I. It is inconstant from month to month, and since it is so irregular, it is valueless as a guide for the normal. The average up to the second year is 1.4 mm. and during the rest of childhood it is 3.5 mm.

T_3 is still more variable. The average for the first two years is one scale division, and for the rest of childhood it is two scale divisions. The average of T_3 for the adult (according to Lewis) is 1.6 mm.

I believe that the height of the T wave is too variable to be used as a standard. It is too easily affected by all external influences. If a baby cried before being cardiographed, or for a moment during the experiment, even though it had no influence on the other deflections, it usually affected the T. That is probably the most plausible reason for the high peaks seen in Figure 6 at periods when there were no physiologic causes; as in Lead I at 4 months and at 1 year; in Lead II at 3 months and at 7 years; in Lead III at 3 months and 6 years.

The T was found inverted rather rarely. It was normal in all leads of all ages, except in Lead III between 6 and 13 years, during which period it was inverted in 15 per cent.

Rate and Rhythm.—There exists a general clinical impression among pediatrists that the pulse of the normal child is more susceptible to changes in rate and rhythm than is that of the adult, that in the new-born, and the infant especially, the pulse is so variable that it is difficult to establish a normal standard, and that arrhythmias occur much more frequently in the new-born and the nursling than in later childhood. I was unable to confirm these opinions by a study of the tracings of hearts of normal children with the electrocardiograph. I found, on the contrary, that the younger the child, the faster is the pulse rate, and that the faster the pulse rate the less frequently arrhythmias occurred. The electrocardiograms in this study were all obtained under exactly similar conditions. In the case of nurslings the readings were taken at the same time of day, right after the feeding, and in older children at about 10 a. m.

The fact that the nurslings were fed before the reading might account for the rather high pulse rates obtained in Tables 5 to 8. The average of from seven to ten pulse periods was taken from each tracing. The time interval for each pulse period was recorded in fiftieths of a second.

Tables 5 to 8, following Krumbhaar, irregularities showing a difference of more than 0.1 of a second, between the shortest and longest

heart cycle, were considered as evidence of sinus arrhythmia. In the new-born I found the average difference between the highest and lowest pulse periods 0.04 second (Table 5). Hecht and Krumbhaar found that at this period the average difference was 0.12 and 0.04 second, respectively. From 6 to 13 years, the figures of Hecht and Krumbhaar are respectively 0.16 and 0.10 second.

TABLE 5.—Pulse Period and Rate of Normal New-Borns

No.	Age	Sex	Weight, Gm.	Pulse Period	Smallest	Largest	Arithmetical Average	Difference	Pulse Rate
1	24 hours	M	3,600	15-15-15-15-15 15-15-15-15-15	15	15	15.0	0	210
2	24 hours	F	2,840	20-20-20-22-20 24-22-20-22-20	20	24	21.0	4	152
3	2 days	M	3,230	35-33-33-30-29 29-29-28	28	35	30.6	7	102
4	2 days	F	2,940	19-20-20-20-20 20-21-21	19	21	20.1	2	159
5	3 days	M	3,250	20-20-19-20-21 20	19	21	16.6	2	158
6	6 days	F	3,440	17-17-17-17-17 17-18-18-18-17	17	18	17.3	1	180
7	5 days	M	3,420	15-15-15-15-15 15-15-15-15-15	15	15	15.0	0	210
8	4 days	M	3,810	19-20-18-18-18 19-18-19-19-19	18	20	18.7	2	158
9	6 days	F	3,440	22-24-23-23-23 24-24-23	22	24	23.2	2	150
10	7 days	F	4,000	23-23-24-25-24 24-24-24	23	25	23.8	2	132
11	7 days	F	3,115	27-25-28-27-27	25	28	26.8	3	118
12	7 days	M	3,430	18-18-18-18-18 18-18-19-18-18	18	19	18.1	1	160
13	11 days	M	3,545	20-19-20-20-19	19	20	19.6	1	158
14	9 days	M	3,900	23-24-25-24-23 23-23-24-25-24	23	25	23.8	2	135
15	9 days	M	3,900	17-18-17-18-18 17-18-17-17-18	17	18	17.5	1	180

Average pulse rate, 157.
Average pulse period, 20.4 (in 1/50 second).
Average difference between highest and lowest pulse period, 1.0 (in 1/50 second).

Although there is a slight difference in the exact time interval in our three studies, nevertheless, the general impression that arrhythmias are more frequent in new-borns than in older children is controverted by all. In general, the pulse rate decreases with age, and with this decrease arrhythmias become more frequent. In the new-born, there was only one case of sinus arrhythmia with a difference of 0.14 second (Table 5, No. 3). In the next group (from 11 days to 1 year) the highest irregularity was 0.10 second (Table 6). In the next group (from 1 to 5 years), there was only one instance in which the differ-

TABLE 6.—Pulse Period and Rate of Normal Nurslings Aged from 11 Days to 1 Year

No.	Age	Sex	Weight, Gm.	Pulse Period	Smallest	Largest	Arithmetical Average	Difference	Pulse Rate
1	6 weeks	M	4,200	19.5-19.5-19.5-19.5-19.5 19.5-19.5-19.5-19.5	19.5	19.5	19.5	0	132
2	6 weeks	M	3,900	21-21-21-22.5-21-21 20-20-20	20.0	22.5	20.8	2.5	144
3	6 weeks	M	4,120	18.7-18.7-18.7-18.7-18.7 18.7-18.7-18.7-18.7-18.7	18.7	18.7	18.7	0	165
4	6 weeks	F	4,100	22.5-23-20.5-21.5-21.5 20.5-22-22-22-24	21.5	24.0	22.0	2.5	144
5	7 weeks	M	4,230	19-19-19-19-19-19-20 20-20-20	19.0	20.0	19.4	1.0	150
6	2 mos.	F	4,330	24-24-24-24-24-24-24 24-24-25	24.0	25.0	24.1	1.0	120
7	2 mos.	F	4,500	26-25.5-25.5-25-25-25 25-23.5	23.5	26.0	25.0	2.5	125
8	2 mos.	M	4,120	23-23-23-23-23	23.0	23.0	23.0	0	135
9	2 mos.	M	4,020	16-16-16-16-16-16-16 16-16-16	16.0	16.0	16.0	0	182
10	7 weeks	F	4,230	20-20-20-20-20-20-20.5 20-20-20	20.0	20.5	20.0	0.5	145
11	74 days	M	3,720	19-20-20-20-20-21.5-24 24-24	19.0	24.0	23.7	5.0	144
12	8 mos.	M	8,100	23.5-23.5-23.5-23.5-23.5 23.5-23.5-23-23-23	23.0	23.5	23.4	0.5	128
13	.....	..	.....	20.5-20.5-20.5-20.5-20.5 20.5-20.5-20.5-20.5-20.5	20.5	20.5	20.5	0	145
14	10 mos.	F	9,000	20-20.5-20.5-20.5-20.5 20.5-20.5-21-21-21	20.5	21.0	20.6	0.5	150
15	11 mos.	M	8,560	26-26-26-26-23.5-28.5 28.5	26.0	28.5	27.0	2.5	117
16	11 mos.	F	8,900	24-24-24-24-24-24-24 24-24-25	24.0	25.0	24.1	1.0	135
17	3½ mos.	F	5,850	24-23-23-23-23-23-24	23.0	24.0	23.2	1.0	132
18	4 mos.	F	6,120	26-26-26-26-26-26-26 26-26	26.0	26.0	26.0	0	118
19	5 mos.	M	4,220	21.5-21.5-21.5-21.5-21.5 21.5-21.5-21.5	21.5	21.5	21.5	0	135
20	5½ mos.	M	4,800	30-29.5-29.5-29.5-29.5 29.5	29.5	30.0	29.6	0.5	126
21	6 mos.	M	5,100	24-22.5-22.5-22.5-22.5-22.5 22.5-22.5-22.5-22.5	22.5	24.0	22.8	1.5	135
22	11 mos.	F	7,650	19-18.8-18.8-18.8-18.8-18.8 18.8-19.5-19.5-19.5	18.8	19.5	19.0	0.7	168
23	11 mos.	F	8,345	27-27-27-27-27-27-27-28.5	27.0	28.5	27.2	1.5	108

Average pulse period, 22.5 (in 1/50 second).
Average pulse rate, 138.
Average difference between highest and lowest pulse period, 1.0 (in 1/50 second).

ence was 0.08 second (Table 7). In schoolchildren, however, (from 6 to 13 years) 47 per cent. showed an arrhythmia over 0.10 second (Table 8).

Transmission Time.—In order to interpret an electrocardiogram correctly, one must first know what constitutes the normal variations in the auricular and ventricular conduction time in children of all ages. In order to establish such a normal standard, I measured the P R interval (auricular activity), the R T interval (ventricular activity) which includes the Q R S and S T intervals, the T P interval (diastolic phase) and the P P interval which represents the complete heart cycle. I then calculated the ratio in the percentages between the three phases of the heart cycle, the P R, the R T, the T P, and the entire heart cycle, the P P.

TABLE 7.—PULSE PERIOD AND RATE OF NORMAL CHILDREN AGED FROM 2 TO 5 YEARS

No.	Age	Sex	Weight, Lbs.	Pulse Period	Smallest	Largest	Arithmetical Average	Difference	Pulse Rate
1	13 mos.	M	21	24–22.5–22.5–21–21–20.5 20.5–20.5–20.5–22.5	20.5	24.0	21.5	4.0	144
2	14 mos.	M	24	31–29–28–30–30–30	28.0	31.0	29.6	3.0	105
3	14 mos.	M	26	22–22–22–22–22–22–22 22–22–22	22.0	22.0	22.0	0	135
4	15 mos.	F	22	28–28–28–28–28–28–27.5 27.5–27.5–26.5	26.5	28.0	27.7	1.5	108
5	15 mos.	F	23	24.5–25.5–25.5–26–27.5 29–25.5–29–29	24.5	29.0	26.6	4.5	108
6	16 mos.	F	24	25–24–29–29–22.5–22.5 22.5–23–24–24	22.5	25.0	23.5	2.5	135
7	19 mos.	F	27	20–20–20–20–20–20–20 20–20–20.5	20.0	20.5	20.0	0.5	145
8	2 years	F	28	15.5–15.5–15.5–15.5–15.5 15.5–15.5–15.5–15.5	15.5	15.5	15.5	0	190
9	2 years	F	26	20–20.5–20–20–20–20.5 20.5–20.5	20.0	20.5	20.3	0.5	120
10	2 years	F	25	21–21–21–21–21–21–21 21–21–20	20.0	21.0	20.9	1.0	144

Average pulse rate, 133.
Average pulse period, 22.7 (in 1/50 second).
Average difference between highest and lowest pulse period, 1.75 (in 1/50 second).

P R Interval.—In the new-born the auricular impulse is shorter than at any later period. It is also remarkably constant, averaging 0.113 second. The longest period was in a baby, 4 days old, 0.15 second, and the shortest 0.09 second. The average obtained from the percentage calculated from each of the tracings is 27.7 per cent. The average obtained from these averages is 26.91, a difference of a little over 1 per cent. (Table 9).

TABLE 8.—Pulse Period and Rate of Normal Children Aged from 6 to 13 Years

No.	Age	Sex	Weight, Lbs.	Pulse Period	Smallest	Largest	Arithmetical Average	Difference	Pulse Rate
1	3½ years	M	35	35-31.5-31.5-33.5-33.5 32-30-31	30.0	35.0	32.2	5.0	85
2	4½ years	M	38	35.5-37-37.5-37	35.5	37.5	36.6	2.0	84
3	6 years	F	42	33.8-32-20-28-28-30-30	28.0	33.8	30.2	5.8	105
4	6 years	M	44	31-30-31.6-33-33-32	30.0	33.0	31.7	3.0	96
5	6 years	F	39	33-36-32-31-30-34-34 34-35	30.0	36.0	33.1	6.0	105
6	6 years	F	37	40-35-31-32-35.5-35.5	31.0	40.0	34.8	9.0	84
7	7 years	M	44	39-40.5-37.5-39-35.5	35.5	40.5	38.5	5.0	82
8	7 years	M	45	33-33-33-33-34-34-33 33-33-33	33.0	34.0	33.2	1.0	96
9	7 years	M	47	34-35-35-41-40-40.5	34.0	41.0	37.6	7.0	84
10	7 years	F	46	32.5-29.5-30.5-34-34 30.5	29.5	34.0	31.7	4.5	96
11	8 years	F	51	36-37.5-37.5-37.5-37.5-37.5 37.5-35.5-37.5-37.5	35.5	37.5	37.1	2.0	99
12	11 years	F	75	27.5-27.5-27.5-27.5-27.5 27.5	27.5	27.5	27.5	0	108
13	8 years	F	50	42-39-42-36.5-44-44.5	36.5	44.5	41.3	8.0	75
14	9 years	M	59	29-31-34-33-30-31.5-37 30.5	29.0	37.0	32.0	8.0	96
15	11 years	M	78	26.5-26.5-26.5-26-26-26	26.0	26.5	26.3	0.5	108
16	11 years	M	76	36-35-36-34.5	34.5	36.0	35.4	1.5	84
17	11 years	M	68	25.5-25.5-25.5-25.5-25.5 26.5-26.5-29-29.5	25.5	29.5	26.5	4.0	115
18	13 years	F	85	29-28-28-28-28-28-28	28.0	29.0	28.1	1.0	108
19	13 years	F	81	30.5-39-35-31.5-31.5-31-35	30.5	39.0	33.3	8.5	80

Average pulse rate, 83.6.
Average pulse period, 33.0 (in 1/50 second).
Average difference between highest and lowest pulse period, 4.3 (in 1/50 second).

TABLE 9.—Transmission Time of Normal New-Borns

No.	Age	P R	R T	T P	P P	Percentage			Pulse Rate
						P R to P P	R T to P P	T P to P P	
1	10 days	0.10	0.23	0.27	0.60	16.6	38.33	45.0	100
2	2 days	0.111	0.21	0.08	0.40	27.5	52.50	20.0	150
3	16 hours	0.12	0.20	0.05	0.37	23.4	54.05	13.5	157
4	24 hours	0.112	0.23	0.05	0.40	30.0	57.5	12.5	152
5	4 days	0.15	0.22	0.08	0.45	33.3	48.88	17.5	135
6	9 days	0.12	0.20	0.03	0.35	34.3	57.14	8.57	174
7	18 hours	0.11	0.19	0.10	0.40	27.5	47.5	25.0	150
8	5 days	0.10	0.21	0.10	0.41	24.3	51.22	24.19	135
9	3 days	0.09	0.21	0.09	0.39	23.3	53.85	23.33	150
Average.....		0.113	0.211	0.94	0.419	27.7	50.07	21.10	144.8
Average percentage calculated from the averages.....						26.9	50.36	22.43	

In the next period (from 11 days to 1 year) the P R interval averages slightly longer (0.125 second). The shortest period is 0.10 and the longest 0.18 second. The P P ratio is 29.34 and 28 per cent., respectively (Table 10). From the first to the fifth year the P R interval is slightly less than in the previous period (0.123), the lowest being 0.10 and the highest 0.16 second. The auricular impulse at this age constitutes from 24.11 to 24.50 per cent. of the entire heart cycle (Table 11).

TABLE 10.—Transmission Time of Normal Nurslings Aged from 11 Days to 1 Year

No.	Age	P Q	Q R S	S T	T P	P P	Percentage				Pulse Rate
							P R to P P	Q R S to P P	S T to P P	T P to P P	
1	4 wks.	0.11	0.03	0.19	0.03	0.36	30.5	8.40	52.77	8.33	164
2	6 wks.	0.13	0.03	0.22	0.07	0.45	28.9	6.67	48.88	15.55	144
3	6 wks.	0.10	0.04	0.21	0.09	0.44	22.7	9.45	47.73	20.45	144
4	6 wks.	0.12	0.04	0.19	0.05	0.40	35.1	4.9	47.5	12.50	157
5	6 wks.	0.12	0.03	0.20	0.05	0.40	32.5	5.00	50.0	12.5	157
6	7 wks.	0.11	0.03	0.21	0.05	0.40	27.5	7.50	52.5	12.5	150
7	11 mos.	0.13	0.04	0.20	0.07	0.42	30.9	4.91	47.62	16.57	135
8	2 mos.	0.12	0.04	0.20	0.08	0.43	27.9	6.99	46.51	18.60	140
9	2 mos.	0.11	0.02	0.18	0.04	0.35	31.4	5.76	51.42	11.42	180
10	4 mos.	0.12	0.02	0.22	0.11	0.47	25.5	4.30	46.8	23.40	132
11	5 mos.	0.15	0.03	0.24	0.04	0.46	32.6	6.54	52.17	8.69	135
12	5½ mos.	0.12	0.04	0.21	0.09	0.48	25.0	12.50	43.75	18.75	126
13	6 mos.	0.13	0.05	0.21	0.04	0.43	30.2	11.67	48.83	9.30	135
14	8 mos.	0.12	0.02	0.21	0.13	0.48	25.0	4.17	43.75	27.08	126
15	10 mos.	0.12	0.04	0.24	0.05	0.45	26.6	8.96	53.33	11.11	135
16	11 mos.	0.18	0.05	0.22	0.10	0.55	38.9	2.92	40.00	18.18	98
17	11 mos.	0.14	0.05	0.27	0.06	0.52	26.9	9.64	51.92	11.54	117
18	14 mos.	0.12	0.04	0.18	0.06	0.40	30.0	10.00	45.00	15.0	150
Average...		0.125	0.036	0.211	0.067	0.44	29.34	7.24	48.36	15.19	133.3
Average percentage calculated from the averages..							28.0	8.18	47.28	15.22	

TABLE 11.—Transmission Time of Normal Children Aged from 1 Year to 5 Years

No.	Age	P Q	Q R S	S T	T P	P P	Percentage				Pulse Rate
							P R to P P	Q R S to P P	S T to P P	T P to P P	
1	13 mos.	0.11	0.04	0.20	0.10	0.45	24.44	8.88	44.44	22.22	135
2	12 mos.	0.11	0.04	0.22	0.11	0.48	22.91	8.33	45.81	22.91	132
3	12 mos.	0.10	0.04	0.22	0.22	0.58	17.24	6.89	37.93	37.93	105
4	14 mos.	0.11	0.07	0.20	0.08	0.46	23.93	15.21	43.47	17.38	125
5	15 mos.	0.12	0.04	0.22	0.20	0.58	20.69	6.89	37.93	34.48	108
6	15 mos.	0.11	0.05	0.25	0.11	0.52	21.15	9.61	48.07	21.15	108
7	16 mos.	0.13	0.04	0.20	0.08	0.45	28.88	8.88	44.44	17.77	135
8	19 mos.	0.11	0.04	0.20	0.20	0.55	20.0	7.27	36.36	36.36	108
9	19 mos.	0.11	0.06	0.20	0.08	0.40	27.5	15.0	50.0	17.5	145
10	2 yrs.	0.12	0.04	0.18	0.08	0.42	28.57	9.52	61.94	19.45	142
11	2 yrs.	0.11	0.06	0.17	0.05	0.39	28.20	15.38	43.59	12.82	158
12	2 yrs.	0.16	0.035	0.22	0.08	0.49	32.65	7.14	44.89	16.32	119
13	3½ yrs.	0.16	0.07	0.23	0.18	0.64	25.0	10.93	35.93	28.12	95
14	4½ yrs.	0.16	0.07	0.28	0.22	0.73	21.92	9.59	38.35	30.13	88
Average...		0.123	0.049	0.213	0.125	0.51	24.50	9.97	43.79	23.19	122.3
Average percentage calculated from the averages..							24.11	9.61	41.76	24.51	

TABLE 12.—Transmission Time of Normal Children Aged from 6 to 13 Years

No.	Age	P Q	Q R S	S T	T P	P P	Percentage				Pulse Rate
							P R to P P	Q R S to P P	S T to P P	T P to P P	
1	6 yrs.	0.12	0.04	0.28	0.11	0.55	21.8	7.27	50.9	20.0	108
2	6 yrs.	0.14	0.06	0.26	0.14	0.60	23.3	10.0	43.3	23.3	100
3	6 yrs.	0.13	0.07	0.30	0.17	0.67	19.4	10.4	44.7	25.3	90
4	7 yrs.	0.16	0.08	0.31	0.18	0.73	21.9	10.9	41.6	24.7	82
5	7 yrs.	0.18	0.08	0.26	0.12	0.64	28.1	12.5	40.6	18.7	95
6	7 yrs.	0.16	0.06	0.26	0.12	0.60	26.6	10.0	43.3	20.0	96
7	7 yrs.	0.14	0.07	0.27	0.09	0.57	24.5	12.2	47.3	15.7	100
8	8 yrs.	0.12	0.08	0.28	0.30	0.78	15.3	10.2	36.2	38.4	72
9	8 yrs.	0.11	0.08	0.28	0.26	0.73	15.0	10.9	38.3	35.6	80
10	9 yrs.	0.12	0.06	0.27	0.22	0.67	17.9	8.9	40.2	35.8	95
11	10 yrs.	0.12	0.07	0.29	0.06	0.54	22.2	12.9	53.5	11.1	105
12	10 yrs.	0.11	0.06	0.29	0.20	0.66	16.6	9.0	43.9	30.3	90
13	11 yrs.	0.14	0.06	0.24	0.07	0.51	27.4	11.7	47.0	13.7	110
14	11 yrs.	0.12	0.08	0.26	0.05	0.51	23.5	15.6	50.9	9.8	112
15	11 yrs.	0.18	0.08	0.26	0.18	0.70	25.7	11.4	37.1	25.7	84
16	12 yrs.	0.15	0.06	0.27	0.06	0.54	27.7	11.1	50.0	11.1	108
17	13 yrs.	0.13	0.08	0.24	0.09	0.54	24.0	14.8	44.4	16.6	108
18	13 yrs.	0.17	0.08	0.30	0.08	0.63	26.9	12.6	47.6	12.6	95
	Average...	0.138	0.069	0.273	0.139	0.620	22.6	11.2	44.4	21.5	961
	Average percentage calculated from the averages..						22.25	11.13	44.03	22.41	

TABLE 13.—Weight of the Musculature of the Left and Right Ventricles in Grams

Boys		Girls		Age	Boys: L.R. Index	Girls: L.R. Index
Left	Right	Left	Right			
3.51	3.8	.....	.....	Fetus 8 mos.	0.92	
5.04	4.35	4.33	3.62	Fetus 9 mos.	1.1	1.1
8.14	6.54	7.95	6.47	Newborn.....	1.2	1.2
12.07	7.23	10.92	6.81	1 to 2 mos.	1.7	1.6
18.13	9.13	16.72	8.61	5 to 6 mos.	2.0	2.0
24.48	12.47	23.49	11.82	11 to 12 mos.	2.0	2.11
29.29	15.92	28.15	14.65	1 to 2 years.....	1.84	1.92
38.51	18.16	36.25	17.69	3 to 4 years.....	2.12	2.05
44.98	23.48	43.07	20.24	5 to 6 years.....	1.97	2.13
49.94	23.33	45.83	23.39	6 to 7 years.....	2.14	1.95
52.25	25.08	48.36	23.39	7 to 8 years.....	2.08	2.06
54.29	26.62	50.76	24.72	8 to 9 years.....	2.04	2.07
57.74	27.7	50.51	26.62	9 to 10 years.....	2.08	1.97
60.39	30.12	59.83	28.54	10 to 11 years.....	2.04	2.10
66.98	32.21	67.3	33.92	11 to 12 years.....	2.79	1.99
72.87	38.0	73.1	36.98	12 to 13 years.....	1.92	1.98
95.45	41.76	96.2	43.7	13 to 14 years.....	2.04	2.13

TABLE 14.—Thickness of the Walls of the Right and Left Ventricles

Author	Age	Right Ventricle	Left Ventricle	L.R. Index
Bednar.....	Fetus 3 mos.	1.5	1.0	0.66
Bednar.....	Fetus 6 mos.	4.0	3.0	0.75
Bednar.....	Birth	0.44–0.68	0.34–0.44	0.75–0.65
Bednar.....	Newborn	4.0	4.0	1.0
Falk.....	Newborn	0.27	0.42	1.55
Gillepsie.....	Newborn	4.0	3.0	0.75
Gillepsie.....	Newborn	5.0	5.0	1.0
Gibson.....	Newborn	4.0	4.0	1.0
Bednar.....	1 month	3.2	5.4	1.68
Gibson.....	1 month	3.2	5.4	1.68
Gibson.....	2–3 months	2.6	6.0	2.31
Gibson.....	4–5 months	3.0	6.5	2.17
Bednar.....	5 months	3.0	6.5	2.17
Falk.....	5–6 months	2.7	5.2	1.92
Gibson.....	6–8 months	2.3	6.8	2.95
Gillepsie.....	11 months	2.0	5.0	2.5
Gibson.....	9–12 months	3.4	8.1	2.38
Gillepsie.....	22 months	2.0	8.0	4.0
Notori.....	2–9 years	3.4–8.1	8.1	2.38
Notori.....	10–20 years	4.0	1.0	2.50

In the last period of childhood (from 6 to 13 years) the P R interval averages 0.138 second. The shortest and the longest intervals are 0.11 and 0.18 second, respectively. The conduction time is from 22.25 to 22.6 per cent. of the entire pulse period (Table 12).

R T Interval.—The R T interval is composed of the Q R S and the S T periods. The Q R S includes from the beginning of Q, wherever present, to the end of S. The S T is from the end of S to the end of T. In the new-born, on account of the frequent absence of the Q, the R T was calculated as a whole. During the first ten

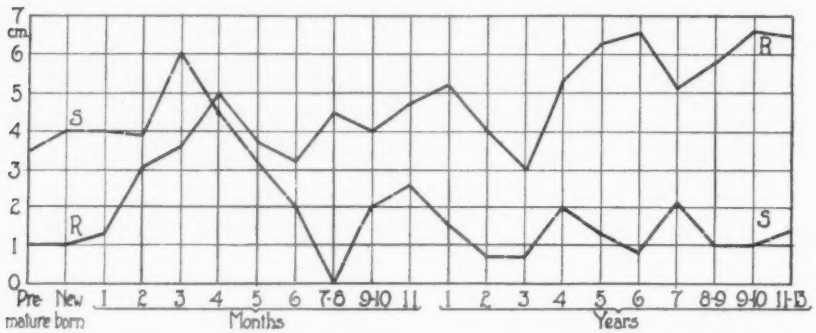

Fig. 7.—Comparison of R_1 and S_1 in all ages. Each square represents 0.5 mlvt. or 5 scale divisions.

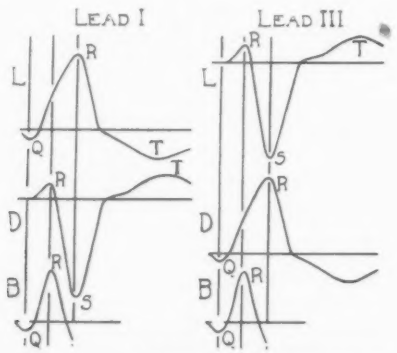

Fig. 8.—Schematic diagram of levogram, dextrogram and bigram (taken from Lewis: Clinical Electrocardiography).

days of life, the period of ventricular activity as thus measured is from 50.07 to 50.36 per cent. of the heart cycle, a little less than twice the auricular activity. It measures 0.211 second, the lowest and the highest being 0.19 and 0.23 second, respectively (Table 9). In the next age (from 11 days to 1 year) the R T phase is somewhat longer. Both the R R S and S T can be recognized, the Q R S averaging 0.036, the S T 0.211, and totaling 0.247 second. The shortest Q R S time is 0.02 and the longest 0.05. It is quite constant in all

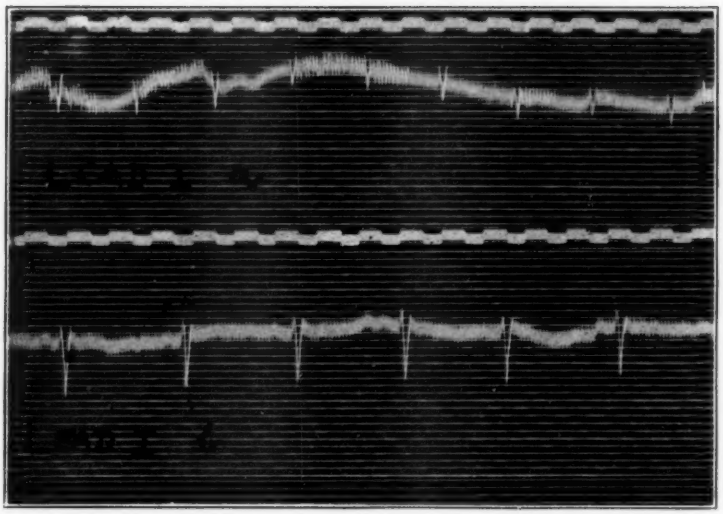

Fig. 9.—Effect of nursing new-born on electrocardiogram: (a) before nursing; (b) after nursing. Lead I only.

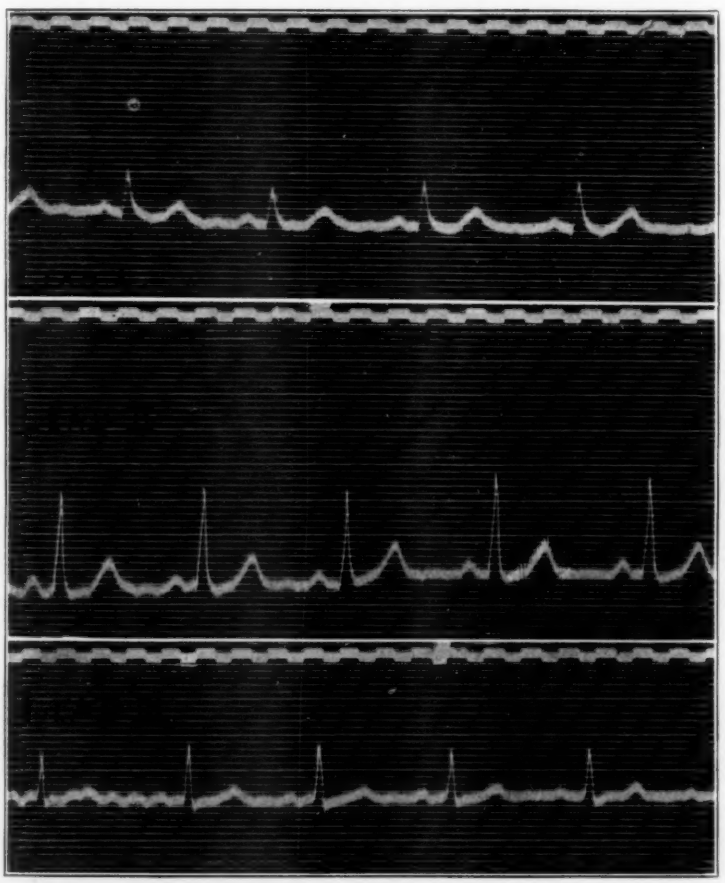

Fig. 10.—Effect on electrocardiogram of change of position: Patient recumbent.

readings. The S T averages 0.211 second. The shortest and the longest intervals are 0.18 and 0.24 second, respectively. The Q R S P P ratio is 7.24 to 8.18 per cent. and the S T : P P ratio is from 47.28 to 48.36 per cent. (Table 10).

In the next period (from 1 to 5 years), the Q R S interval averages 0.049 second and ranges from 0.035 to 0.07 second. It comprises from 9.61 to 9.97 per cent. of the entire heart cycle.

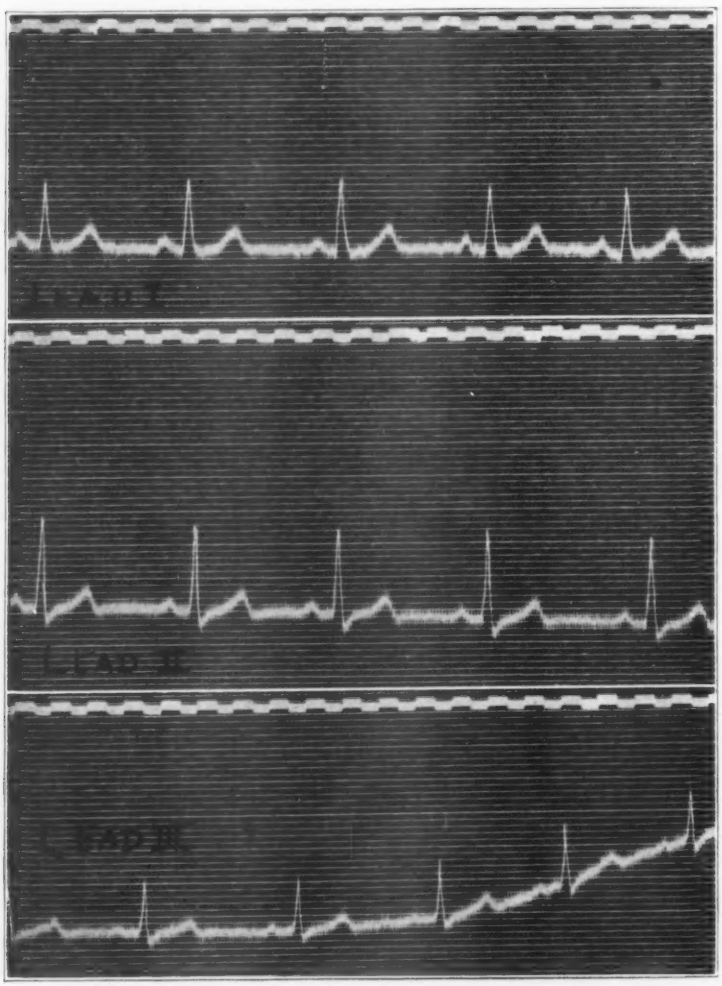

Fig. 11.—Effect on electrocardiogram of change of position: Patient seated.

The S T averages 0.213 second, and ranges from 0.17 to 0.28 second. It is from 41.76 to 43.79 per cent. of the pulse period. The total Q T interval during this period averages 0.262 and is about 51 per cent. of the pulse period (Table 11).

The Q S interval during the latter part of childhood averages 0.069 second and ranges from 0.04 to 0.08 second. The S T averages 0.273 second and ranges from 0.24 to 0.31 second. The Q R S : P P ratio is 11.2 per cent., and the S T : P P ratio is 44.2. per cent. The total Q T interval averages 0.342 second and the total Q T : P P ratio is 55 per cent. The average Q T in the adult is 0.36 second (Q R S 10) and (S T 0.26 second) (Table 12). With the exception of the S T during the latter part of childhood, the duration of both auricular and ventricular conduction time is less than in the adult.

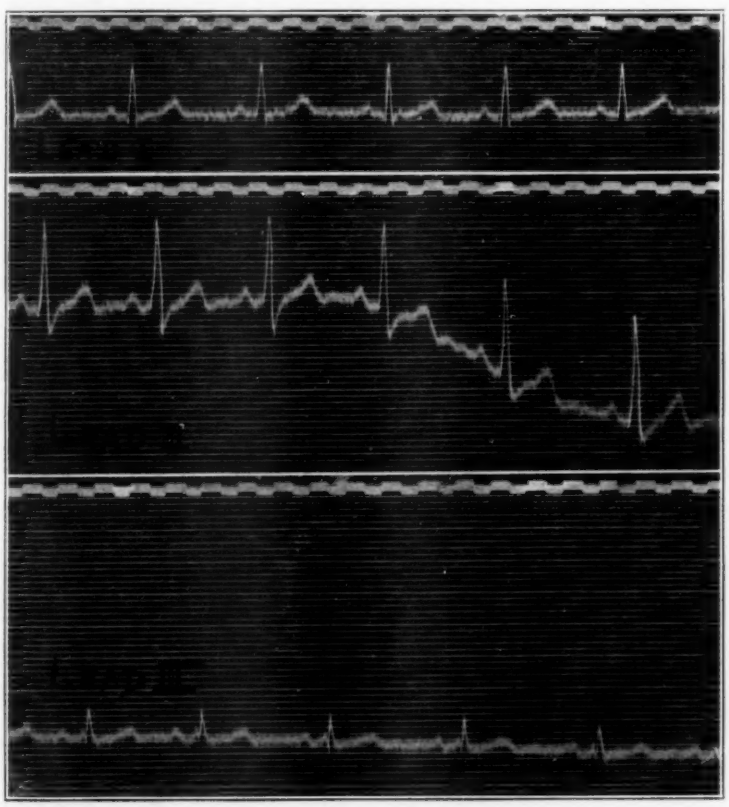

Fig. 12.—Effect of change of position on electrocardiogram: Patient lying on right side.

T P Interval.—The T P interval represents the period of rest. Obviously it depends on the pulse rate, and is so variable and inconstant that it is useless as a guide for the normal.

Ventricular Preponderance.—Lewis [23] defines ventricular preponderance as the disturbed weight ratio between the two ventricles. This may be due to pathologic changes, as in heart disease, or it may be an entirely physiologic phenomenon, as in the normal growth of the

heart during and through childhood. It is difficult many times, particularly in the infant, to decide clinically whether a hypertrophy exists. In such an instance the electrocardiograph is especially valuable.

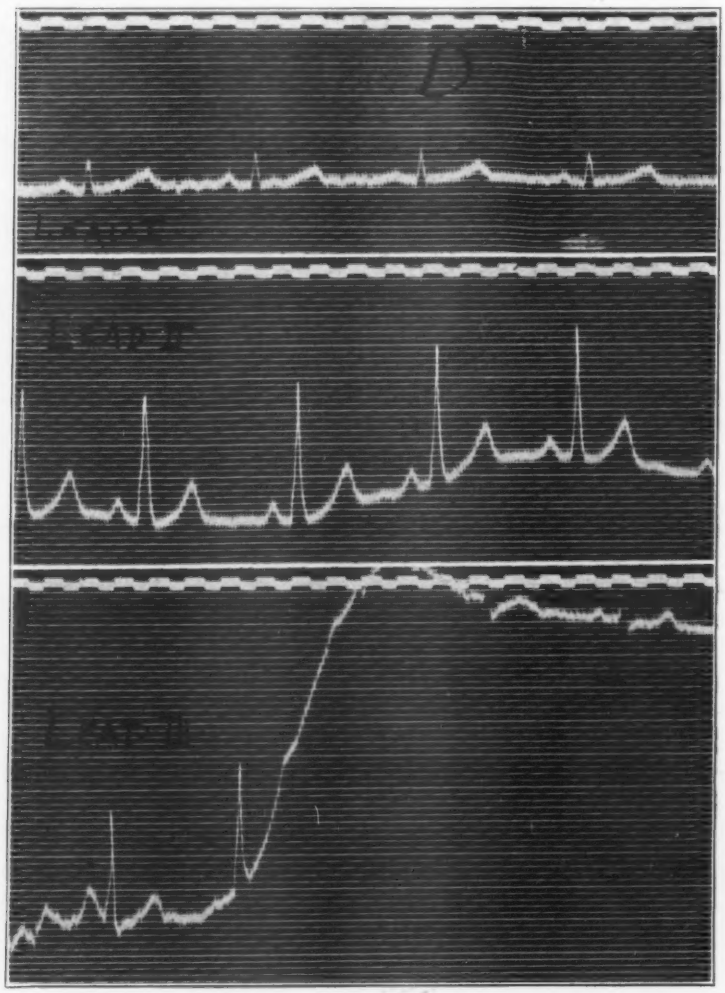

Fig. 13.—Effect of change of position on electrocardiogram: Patient lying on left side.

Figure 8 (taken from Lewis) is a schematic diagram of the separate curves of each ventricular phase in each lead. In the upper curve (1) the right division of the bundle which transmits impulses through the ventricle is severed below its origin, and the normal impulse no longer travels through it but instead spreads itself through the left branch. Lewis terms this levogram.

The middle curve (D), termed the dextrogram, represents the opposite condition in which the impulse passes only through the right ventricle branch and the left ventricle branch is severed. The lowest curve is the "bigram" and represents the algebraic sum of the other two.

All observers are agreed that in the premature, in the new-born, and for a short time thereafter, there is a definite and characteristic

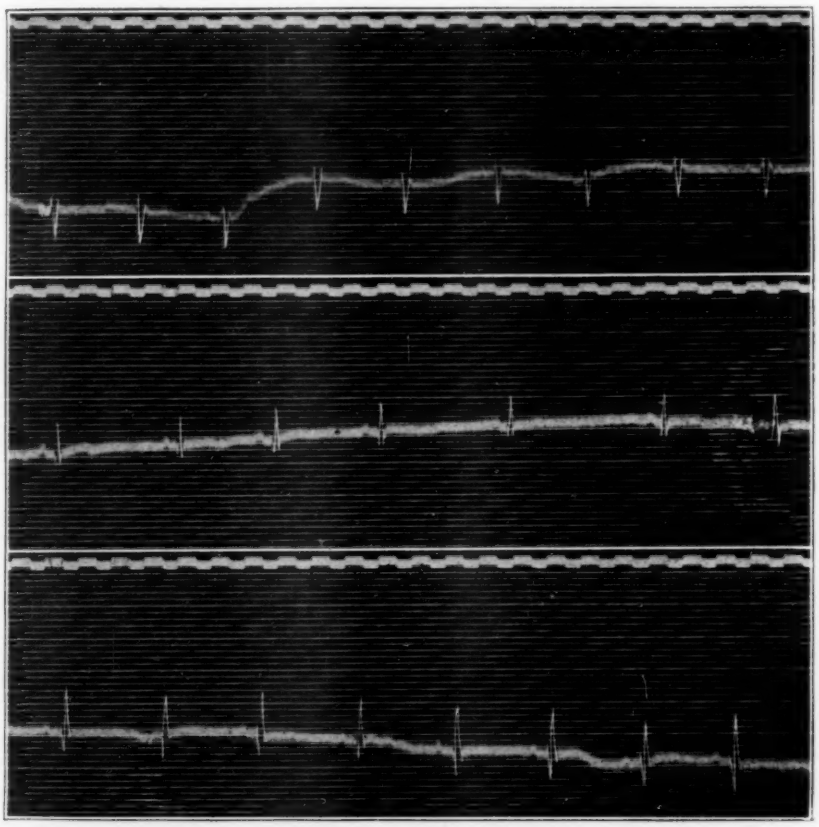

Fig. 14.—Electrocardiogram of normal new-born 24 hours old.

electrocardiogram which in form and height of deflection is entirely different from that of the older child and that of the adult. Funaro, in 1908, under the direction of Nicolai, first observed this cardiogram. He concluded that it represented a left ventricular preponderance. The anatomic data in our possession at the present time contradict this opinion. Heubner, Noeggerath [14] and others also noted this peculiar type of curve in the new-born. They explained it by the high position of the diaphragm, and consequently the more horizontal axis of the

heart. This theory could not be confirmed by the electrocardiograms which I obtained from subjects lying either on their left or right sides. In these instances although the height of the deflections changed in the different positions the outline of the curve remained essentially the same (Figs. 9, 10, 11, 12 and 13).

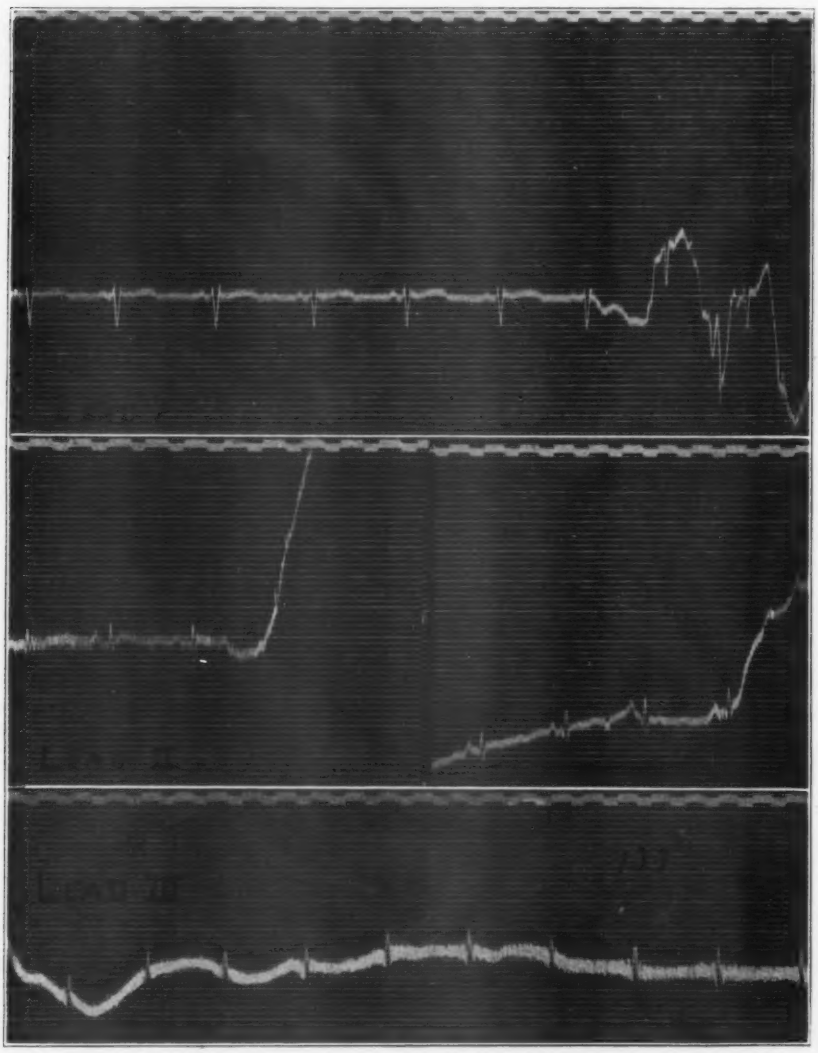

Fig. 15.—Electrocardiogram of normal new-born 48 hours old.

What effect do the muscle mass, the thickness of the walls, and the weight of the ventricle have on the electrocardiogram? A careful study (Tables 13 and 14) of the anatomic data obtained from dis-

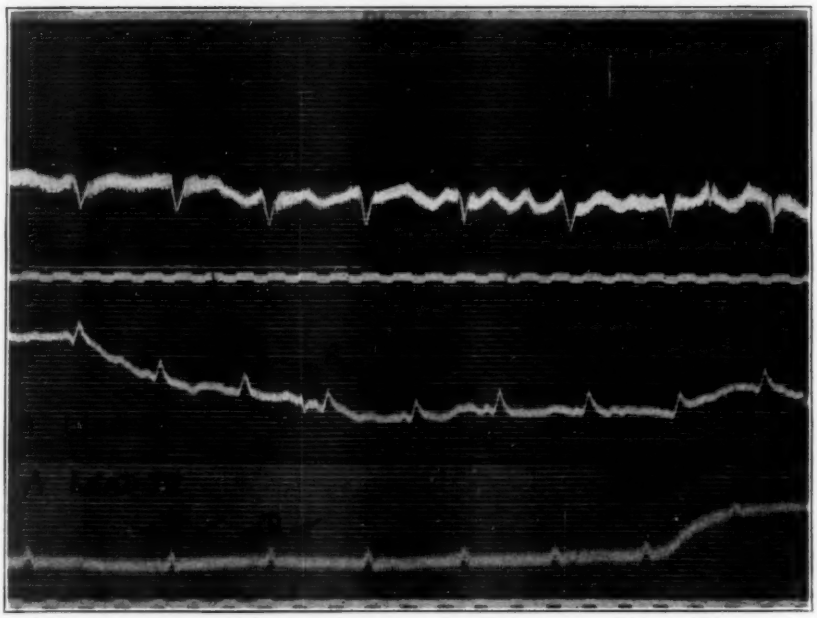

Fig. 16.—Electrocardiogram of a new-born 4 hours old.

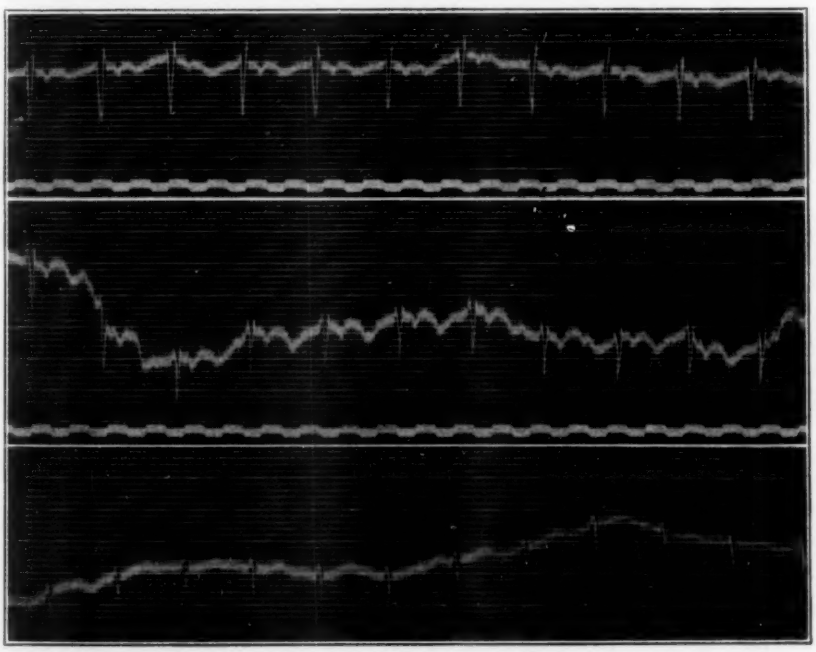

Fig. 17.—Electrocardiogram of the same new-born (Fig. 16) 4 weeks old.

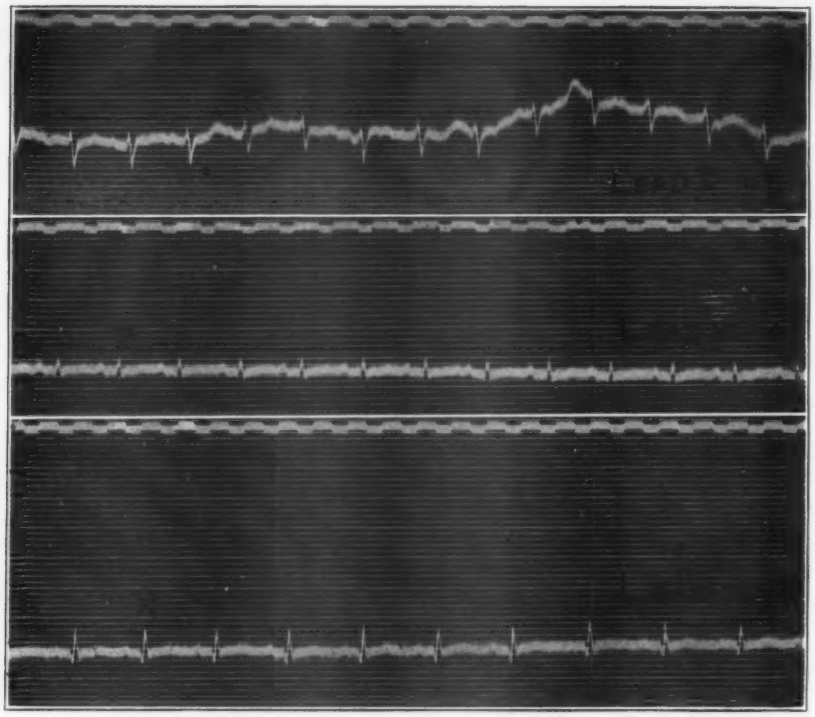

Fig. 18.—Electrocardiogram of new-born 5 days old.

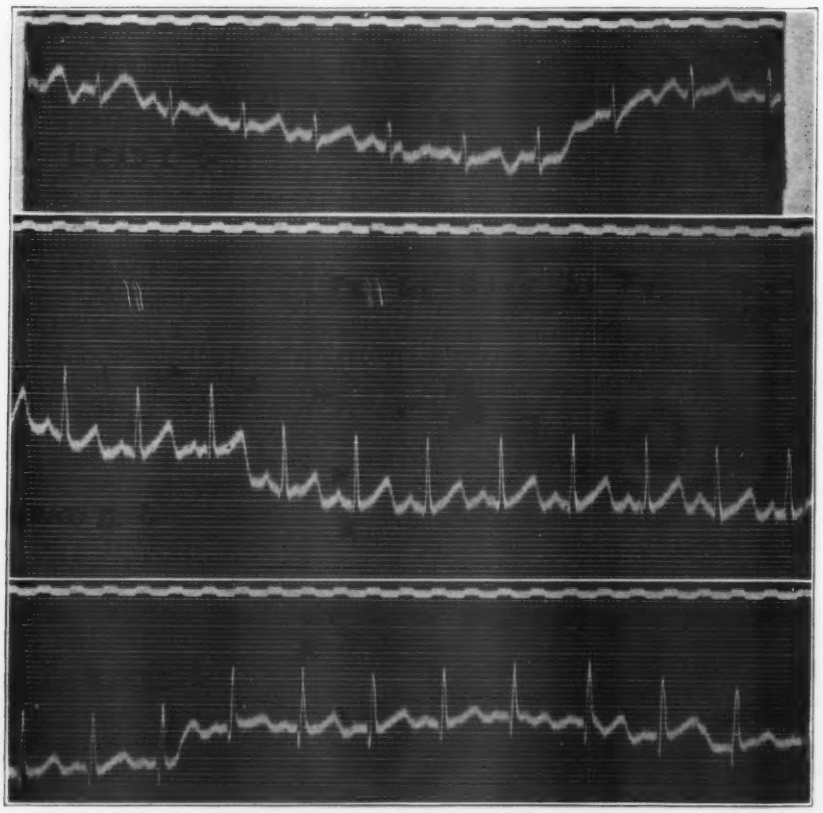

Fig. 19.—Electrocardiogram of the same new-born (Fig. 18) 6 months old.

sections of hearts of children of all ages shows that there is a definite right ventricular preponderance in the new-born and during the first two to three months; that, whereas in the adult the left ventricle weighs from 1.8 (Lewis) to 2.6 (Pfaundler) as much as the right ventricle, in the new-born the two ventricles are about equal. At birth the walls of the right ventricle are of about the same thickness as those of the left ventricle, slightly less. In other words, the L/R index of the thickness of the walls is from 0.66 to 1.0, and the L/R index of the weight is approximately 1.0.

Taking the adult electrocardiogram as a standard for comparison, it can be seen why that of the new-born shows a right ventricular preponderance, curve S 1 and 2. In this curve, the S is exaggerated in Lead I and shorter in Lead III. The R has very little or no amplitude in Lead I and is highest in Lead III. The Q is higher at this age in Lead II and III than in the adult. It should be mentioned, however, that the above anatomic figures are not entirely criticism proof; first, because there is insufficient material; secondly, because the measurements have been taken at different heights by different authors, and thirdly, because some of the cases reported are probably still-births, which fact presupposes disease or prematurity.

CONCLUSIONS

1. In the premature and during the first three months of life, the normal electrocardiogram indicates a right ventricular preponderance. At about the fourth month, the R_1 becomes larger than the S_1, and from then on the ventricular complex approaches the adult type of curve.

2. In general, the average heights of the deflections in childhood are greater than in the adult.

3. Except in the premature, satisfactory records showing all the deflections seen in the normal adult electrocardiogram can be obtained during any period of childhood.

4. The P is higher in childhood than in the adult, in all three leads. Except in the premature, it was absent in my series of cases in only two instances. It was found electronegative in eight records.

5. The Q plays a prominent part in the electrocardiogram of childhood. It is larger than in the adult, especially in Leads II and III. Altogether it adds confirmatory evidence that there is a right ventricular preponderance in the first few months of life.

6. The R in Lead I during the first few months of life is lower than at any other period, but in Leads II and III it averages higher than in the adult. The increase of R_1 corresponding with the decrease of S in Lead I is also indicative of a right ventricular preponderance during the earliest periods of life.

7. The S deflection is the most characteristic and distinctive of the ventricular complex in childhood. In Lead I it is both relatively and absolutely higher in the first three months than in any other period of life and in the other leads it is relatively higher than in the adult.

8. The T is very susceptible to external influences in the child, and therefore its height is unreliable. In the first ten days of life it is quite frequently absent. After that it is quite constant. It was found inverted, but only during the latter part of childhood, in 15 per cent. of the readings.

9. The general impression that the younger the child the more frequent the arrhythmias was not confirmed by the electrocardiograph. The younger the child, the faster the pulse; and the faster the pulse the less frequent the arrhythmias. In the new-born the average difference between the highest and lowest pulse periods was $\frac{2}{50}$ second, whereas in the older children (from 6 to 13 years) the average difference was $4\frac{3}{50}$ seconds. In the period of infancy, no sinus arrhythmia occurred (difference of 0.1 second or more) while in the school age 47 per cent. showed sinus arrhythmias.

10. The transmission time in children is, on the whole, of shorter duration than in the adult. In the new-born the auricular activity occupies an average of 0.113 second, whereas, in the last period of childhood it consumes an average of 0.138 second. In the adult the P R interval averages 0.18 second.

11. The ventricular conduction time in the child corresponds to the auricular. In the new-born it averages 0.21 second and gradually increases until at the school age it averages 0.34 second. In the adult the average for the R T period is 0.36 second.

12. The right ventricular preponderance which characterizes the first few months of life can best be explained by the relationship of muscle mass between the right and left ventricles.[14]

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PHYSICAL DEFECTS IN CHILDREN

REPORT OF SIX HUNDRED AND TWO CASES *

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In an article published in 1917,[1] a group study was presented showing the results of one hundred physical and mental examinations of so-called well children made at the Little Wanderers' Home in Boston. This report includes the children in the former study, together with 145 additional examinations made at the same institution, and 357 made in the Children's Out-Patient Department of the Massachusetts General Hospital.

One of the objects of the investigation has been to emphasize the importance of more systematic and thorough methods of physical examination for all children. A standard form has been worked out in our nutrition clinics,[2] which covers all the practical points in examination in regular order; second, enables different examiners to cover the same ground; third, saves clerical work by so arranging descriptive matter as to allow underlining instead of writing; fourth, acts as a check on the work of the examiner, since a failure to underline an item shows failure to examine.

I. DEFECTS FOUND

The children examined in the Little Wanderers' Home exhibited a larger proportion of defects than would be found in an ordinary school, yet each of these children had already been given a routine examination and declared to be sufficiently well to attend school and to engage in the ordinary activities of normal children. The other and larger group is made up of the usual type of child met in the out-patient department of a large hospital.

The advantages of more adequate examinations of children in both these classes are evident. In the hospital, there is great waste of time and energy due to the result of failure to make complete initial examinations and records. The value to those concerned with members of "the so-called well" group appears in the case of O. B., a boy aged 7,

* Received for publication, Nov. 15, 1920.

1. A group study of the results of one hundred physical and mental examinations of so-called well children. Internat. Clin. **2**: Series 27, 1917.

2. A copy of this form for history and physical examination, with other blanks and forms used in our nutrition clinics, will be found in an article on malnutrition in children, a class clinic, in Internat. Clin. **4**: Series 29, 1919.

who, after having been reported normal, was found to require care for ten defects: (1) 15 per cent. underweight for height; (2) adenoids; (3) hypertrophic pharyngitis; (4) diseased tonsils; (5) five carious teeth; (6) enlarged cervical glands; (7) cerumen in both ears; (8) round shoulders; (9) adherent prepuce; (10) mouth breathing. This was one of the worst cases found, yet Table 1 shows that twelve children, or 5 per cent. of the group to which this boy belonged, had ten or more defects. The distribution of defects in this group, according to number, is shown in Table 1.

TABLE 1.—NUMBER OF DEFECTS PER INDIVIDUAL AMONG SIX HUNDRED AND TWO EXAMINED

Number of Defects	Little Wanderers' Home, 245 Cases	Massachusetts General Hospital, 357 Cases
None	4	5
One	8	2
Two	24	5
Three	34	12
Four	36	26
Five	33	32
Six	32	54
Seven	31	78
Eight	24	85
Nine	7	35
Ten	9	12
Eleven	2	7
Twelve	1	4

It will be noted that the mode—the largest number of cases—is the group of four defects in the Little Wanderers' Home class and that of eight in the Massachusetts General Hospital class. The average number of defects found in the former was 5.2, and in the latter, 6.8. The larger number resulting from the Massachusetts General Hospital examinations is due, in great part, to two causes: first, as stated above, these latter children were candidates for clinic care, and so, elite, represented a lower grade of physical condition; second, these examinations were conducted later than those in the Little Wanderers' Home class when our method of examination and record had been so worked out that fewer defects escaped observation.

The children represented all stages of the school and preschool periods, but the largest number in each institution were from 7 to 9 years of age. Table 2 shows that at least a majority of physical defects were present as early as the preschool period.

Table 2 shows a remarkable uniformity in defects at various ages. There is no evidence of any particular age at which children have an especially large number of defects.

Because we have found obstruction to breathing to be a serious matter as regards growth and development, we have made a special

grouping of nasopharyngeal defects. Here, again, we find remarkable uniformity at various ages.

TABLE 2.—AVERAGE NUMBER OF PHYSICAL DEFECTS AT VARIOUS AGES

Age	Little Wanderers' Home			Massachusetts General Hospital		
	Per Cent. of Total Group	Average Number Defects		Per Cent. of Total Group	Average Number Defects	
		All Kinds	Naso-pharyngeal		All Kinds	Naso-pharyngeal
3 and under.....	12	5.0	3.9	9	6.0	3.6
4–6.....	21	5.0	2.5	23	6.9	3.5
7–9.....	23	5.1	2.3	30	7.2	3.6
10–12.....	18	6.0	3.3	28	6.9	3.8
13 and over.....	17	4.3	2.4	6	6.0	3.0
Unknown.....	9	3.5	1.3	4	6.4	3.5
Entire group.....	100	5.2	2.5	100	6.8	3.5

Table 3 gives a summary of the most general facts brought out in the examinations. These are grouped in order to show, first, the number of individual cases found suffering from certain defects; and, second, the actual number of defects found. For convenience the results are stated in the four classes which it has seemed most important to distinguish in order to study their incidence—underweight, naso-pharyngeal obstruction, carious teeth, and defects of posture.

TABLE 3.—SUMMARY SHOWING NUMBER OF INDIVIDUALS HAVING DEFECTS AND NUMBER OF DEFECTS

Cases Suffering from Defects	Little Wanderers' Home						Massachusetts General Hospital					
	Male		Female		Both Sexes		Male		Female		Both Sexes	
	Cases	Per Cent.	Cases	Per Cent.	Cases	Per Cent.	Cases	Per Cent.	Cases	Per Cent.	Cases	Per Cent.
Number of physical examinations made..	160	100	85	100	245	100	163	100	194	100	357	100
Number found not needing treatment...	2	...	2	...	4	...	3	...	2	...	5	...
Cases of nasopharyngeal obstruction.....	140	88	73	86	213	87	159	98	185	95	344	96
Cases of teeth defects.	95	59	48	56	143	58	115	71	138	71	253	71
Cases of postural defects.....	83	52	38	45	121	50	109	67	142	73	251	70
Actual Number of Defects Found	**De-fects**	**Per Cent.**	**De-fects**	**Per Cent.**	**De-fects**	**Per Cent.**	**De-fects**	**Per Cent.**	**De-fects**	**Per Cent.**	**De-fects**	**Per Cent.**
Underweight for Ht.* 10% and over.....	43	27	23	27	66	27	95	58	127	66	222	62
7% and over.....	...	...	...	...	...	...	118	72	150	77	268	75
Nasopharyngeal.....	393	246	224	263	617	252	593	364	656	338	1249	350
Teeth.....	114	71	50	59	164	67	129	79	147	76	276	78
Postural.....	118	74	56	66	174	71	145	89	201	106	346	97
Other.....	146	91	96	113	242	100	141	86	143	74	284	80
Total basis 10% underweight.....	814	509	449	528	1263	517	1108	676	1274	660	2377	667
Total basis 7% underweight.....	....	....	....	....	....	....	1126	690	1297	671	2423	680
Number of cases with mental defects.....	58	36	23	27	81	33						

* In underweight for height the number of cases and the number of defects is, of course, the same.

Table 4 presents the remaining defects found. These are not used in the incidence tables, but should be useful in making comparisons with the results of other examinations.

In Table 5 will be found a comparison between the results we have secured in the two institutions and those which appear in examinations made in the course of school medical inspection in two of our largest cities.

TABLE 4.—SUMMARY OF "OTHER" DEFECTS NOT PRESENTED IN INCIDENCE TABLES.

	Little Wanderers' Home			Massachusetts General Hospital		
	Male	Female	Both	Male	Female	Both
Eyes	7	4	11	5	8	13
Ears:						
Cerumen and foreign bodies	41	25	66	25	36	61
Otitis media*	14	13	27	7	6	13
Thickened drum*	0	0	0	87	82	169
	—55	—38	—93	—119	—124	—243
Cardiac	2	1	3	2	6	8
Lungs	3	4	7	8	4	12
Skin	13	15	28	13	21	34
Genito-urinary system	57	7	64	41	5	46
Enuresis	9	10	19	11	8	19
Pediculosis	10	22	32	19	43	62
Nervous system	1	1	2	5	1	6
Overweight	0	1	1	1	10	11
Anemia	2	1	3	1	0	1
Cleft palate	0	0	0	1	0	1
Periosteitis	1	5	6	0	0	0
Tuberculosis	0	0	0	6	1	7
Infantile paralysis	0	0	0	1	0	1
Measles	0	0	0	1	0	1
Pertussis	0	0	0	1	0	1

* Otitis media and thickened drum are also included in nasopharyngeal defects.

TABLE 5.—COMPARISON BETWEEN THE RESULTS PRESENTED IN THIS STUDY AND THOSE FOUND IN TWO REPRESENTATIVE CITY SCHOOL MEDICAL INSPECTIONS

Defects	Little Wanderers' Home, per Cent.	Massachusetts General Hospital, per Cent.	1919 New York, per Cent.	1918 Philadelphia, per Cent.
Number examined	245	357	248,978	147,631
Vision	4.5	3.6	7.2	13.5
Hearing	38.0	69.0	0.5	9.8
Nasopharyngeal	252.0	350.0	26.8	16.2
Defective nutrition	27.0	75.0	20.0	11.7
Cardiac	1.2	2.2	1.6	0.6
Pulmonary	2.8	3.4	0.3	
Orthopedic	64.0	90.0	0.9	15.5
Nervous system	0.8	2.0	0.6	0.3
Teeth	67.0	77.0	62.0	48.0
Skin	11.5	9.5	....	6.5
Pediculosis	13.0	17.5	....	4.1
Enuresis	7.7	5.3	....	0.015
Genito-urinary system	26.0	13.0		
Stoop shoulders	42.0	65.0	....	1.3
Lateral curvature	15.0	22.0	....	0.13
Flat foot	6.0	9.0	....	0.006

Comparison with Results of Other Studies.—Little attempt has been made to compare the results secured in these examinations with those coming from any other studies. The difficulties in the way of such

comparisons are due, principally, to two causes: first, the difference in attitude and approach of various physicians making the examinations; second, the varying physical conditions found among different groups of children.

The first of these will be remedied as fast as it is possible to standardize examinations along uniform lines. The second difficulty is similar to that found in various studies made with reference to the apparent increase of certain diseases among adults. Changes in type of population and other factors must be carefully investigated before generalizations can be made. This wide variation is illustrated in a study made in New York City of about 2,500 school children. It was found that these children showed an average of 49 per cent. of teeth defects and 61 per cent. of tonsil defects. But an analysis of these figures showed that in teeth defects the boys averaged 45 per cent. and the girls 53 per cent.; in tonsil defects the boys averaged 54 per cent. and the girls 70 per cent. A further analysis among the boys showed that the percentage in teeth defects for the foreign born was 36, for the native born, 48; in tonsil defects the foreign born averaged 46 per cent. and the native born 52 per cent.

When the various nationalities were separated, generalization became even more difficult, as shown in Table 6.

TABLE 6.—THE DISTRIBUTION OF TEETH AND TONSIL DEFECTS ACCORDING TO NATIONALITY *

	United States, per Cent.	Italy		Russia, Poland		Germany, Austria		Hebrew	
		Foreign Born, per Cent.	Native Born, per Cent.	Foreign Born, per Cent.	Native Born, per Cent.	Foreign Born, per Cent.	Native Born, per Cent.	Foreign Born, per Cent.	Native Born, per Cent.
Teeth..	52	37	43	59	40	24	48	30	56
Tonsils	50	49	55	63	39	37	53	42	54

* A New York City study made by the Association for Improving the Condition of the Poor.

Number of Defects Found by Different Examiners.—The differences in the defects found in examinations made by various physicians were studied at the same time. Two hundred and sixty-six children were examined consecutively but independently by each of three physicians who were found to agree in only 75 per cent. of the cases with reference to the presence or absence of carious teeth, and in 63 per cent. with reference to the presence or absence of diseased tonsils.

Relationship of School and Growth Records to Defects.—In this New York study, a further comparison was made of the correlation found between teeth and tonsil defects, on the one hand, and, on the other, school attendance and promotions, also height and weight.

School attendance was based on the actual records for an entire year. Promotions were studied with reference to the changes made in school grade by each child during the same period of time. Height and weight were based on the relationship of each child to the Boas-Burk averages for his age.

The first line in Table 7, under boys, would be read: "Boys having no teeth defects showed over those having such defects 7 per cent. better attendance in school, made 6 per cent. more promotions, showed no difference in height for their age, and 7 per cent. more weight for their age."

TABLE 7.—STUDY OF 1,242 SCHOOLCHILDREN WITH REFERENCE TO THE RELATION OF TEETH AND TONSIL DEFECTS TO SCHOOL ATTENDANCE, PROMOTION, HEIGHT AND WEIGHT (A. I. C. P., NEW YORK)

	School Attendance, per Cent.	School Promotions, per Cent.	Height, per Cent.	Weight, per Cent.
Boys:				
No teeth defects	7	6	0	7
No tonsil defects	7	1	6	7
Neither teeth nor tonsil defects	10	8	5	8
Girls:				
No teeth defects	..	3	4	15
No tonsil defects	..	3	6	14
Neither teeth nor tonsil defects	..	15	14	25

TABLE 8.—COMPARISON OF THE RESULTS OF THE EXAMINATION OF FIFTY CHILDREN * (BOTH SEXES) AT A CHILDREN'S CLINIC AND AT THE NUTRITION CLINIC

Defects Found	(1) Defects Appearing in Diagnosis Summary	(2) Additional Defects Shown in Various Parts of Record	(3) Total of (1) and (2)	(4) Defects Found in Nutrition Clinic	(5) Per Cent. (3) of (4)	(6) Per Cent. (1) of (4)
Cases exam.	50	50	50	50	100	100
All defects:						
Cases	42	38	50	50		
Defects	83	93	176	401	44	20.8
Nasopharyngeal:						
Cases	12	28	32	50		
Defects	17	40	57	191	30	8.9
Teeth:						
Cases	15	17	32	32	100	47
Defects	.	..	..	150		
Postural:						
Cases	10	8	18	50		
Defects	11	9	20	108	18.5	10.2
Underweight:						
Cases	11	21	32	39	82	28

* Average age about 9 years.

Table 8 presents some very interesting data showing the defects found in fifty children who were patients in one of our largest and best organized children's clinics, as compared with the results of the

examinations of the same children made in our nutrition clinic. The figures are given in four columns, showing, first, the defects appearing in the diagnosis summary; second, the additional facts scattered through the cards filed in the case; third, the total of (1) and (2); and, fourth, the defects found in the examination made at the nutrition clinic. The fifth and sixth columns show the percentage comparisons. The general examination found only 44 per cent. of the number of defects appearing in the records of the examination at our nutrition clinic, and in the various groups, from 18.5 per cent. in defects of posture to 100 per cent. in teeth defects.

It would be more reasonable (column 6) to compare the summary of defects made in the nutrition clinic examinations with the first column giving the summary in the other situation. This greatly decreases the percentages shown in the fifth column, for only 20.8 per cent. of all defects found in the nutrition examination appear in the diagnosis summary, ranging from 8.9 per cent. of nasopharyngeal obstructions to 47 per cent. of teeth defects. If the results of an examination are not clearly stated in the diagnosis summary, they are usually overlooked, as treatment is rarely ordered except for defects thus stated.

It might be urged that the general examination is especially concerned with the vital organs, and omits only the less essential defects, but an inspection of the results of the more complete examination shows that no defects have been included which do not have direct bearing on the health of the child.

II. INCIDENCE OF DEFECTS

The larger groups in this study have been analyzed in order to find the correlation of defects. These groups include underweight, nasopharyngeal obstructions, teeth defects, and postural defects. In the last named group, further analysis has been made of round shoulders, lateral curvature and flat foot.

The comparisons in each section are between those individuals showing the particular defect and those who were free from it, as well as with the total group including both.

Nasopharyngeal Obstruction.—Table 9 shows the various nasopharyngeal obstructions found as well as their distribution in the children from the two institutions studied:

In Table 10 is indicated the percentage of postural, underweight and teeth defects occurring in the nasopharyngeal group, as compared with those who were free from these obstructions, as well as with the

entire membership in both sections of the study. It will be recalled that only 13 per cent. of those examined at the Little Wanderers' Home, and 4 per cent. of those at the Massachusetts General Hospital, were found to be free from nasopharyngeal defects.

TABLE 9.—DISTRIBUTION OF NASOPHARYNGEAL OBSTRUCTION

Defects	Little Wanderers' Home			Massachusets General Hospital		
	Male, per Cent.	Female, per Cent.	Both, per Cent.	Male, per Cent.	Female, per Cent.	Both, per Cent.
Adenoids	62	65	63	29	21	25
Hypertrophic pharyngitis	42	46	44	77	78	77
Diseased tonsils	50	55	52	67	60	63
Deviated septum	15	12	14	10	5	7.5
Spur	5	2.4	4	2.5	1.5	2
Hypertrophic turbinates	17	20	17.5	1	3	2.2
Open mouth	18.5	30	23	59	59	59
Enlarged anterior cervical glands	26	20	24	61	65	63
Otitis media	9	15	11	4	3	3.6
Thickened drum	0	0	0	53	42	47
Total	244.5	265.4	252.5	363.5	337.5	349.3

TABLE 10.—INCIDENCE OF POSTURAL, TEETH, AND UNDERWEIGHT DEFECTS IN THE NASOPHARYNGEAL GROUP

Defects	Nasopharyngeal Defect								
	Male			Female			Both Sexes		
	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.
Little Wanderers' Home									
Postural	54	38	52	47	31	44	52	35	49
Teeth	59	66	59	60	33	56	59	53	58
Underweight	26	29	26	26	31	27	26	29	26
Mass. General Hospital									
Postural	67	67	67	74	50	73	71	54	70
Teeth	71	33	71	74	30	71	73	31	71
Underweight	73	67	73	77	70	77	75	60	75

The comparison shows that among the girls, those having nasopharyngeal defects indicate for the Little Wanderers' Home and Massachusetts General Hospital sections 16 and 24 per cent., respectively, more postural defects than occur among those free from these obstructions. A like disadvantage in this respect appears among the boys of the former institution. In like manner, three out of four sex divisions show from 27 to 44 per cent. more teeth defects than appear in the cases free from nasopharyngeal obstruction. In percentage of defects from underweight for height, the Little Wanderers' Home children show a negative correlation, but those in the Massachusetts General Hospital section show a positive relation of 6 and 7 per cent.

Defects of the Teeth.—The lack of correlation between malnutrition and carious teeth is remarkable. There seems to be no evidence that small cavities in teeth affect nutrition unless there are also abscesses or other inflammatory conditions. Below are given the distribution of cases and of defects.

TABLE 11.—DISTRIBUTION OF DEFECTS OF TEETH

Defects	Little Wanderers' Home			Massachusetts General Hospital		
	Male Cases	Female Cases	Both Sexes Cases	Male Cases	Female Cases	Both Sexes Cases
Carious teeth	89	47	136	118	138	256
Approximation	6	1	7	2	0	2
Alveolar abscess	5	0	5	5	4	9
Gingivitis	1	1	2	1	0	1
Pyorrhea	0	0	0	0	1	1
Stomatitis	0	1	1	0	1	1
Total cases	95	48	143	118	138	256
Per cent.	59	56	58	72	71	72
Total defects	101	50	151	126	144	270
Per Cent.	63	59	62	77	75	76

In Table 12 the most evident correlations are found between teeth and postural defects, ranging from 7 to 22 per cent. of the latter among children suffering from the former. Three of the four divisions on the basis of sex show from 4 to 16 per cent. greater incidence of nasopharyngeal obstruction among those having teeth defects. Only one of the four divisions shows a positive correlation between teeth defects and underweight.

TABLE 12.—INCIDENCE OF NASOPHARYNGEAL, POSTURAL, AND UNDERWEIGHT DEFECTS WITH REFERENCE TO DEFECTS OF THE TEETH

Defects	Teeth Defects								
	Male			Female			Both Sexes		
	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.
Little Wanderers' Home									
Nasopharyngeal	85	86	86	92	76	85	87	83	85
Postural	57	44	54	54	32	44	56	39	49
Underweight	24	29	26	23	32	27	24	30	26
Mass. General Hospital									
Nasopharyngeal	100	96	98	97	89	95	98	92	96
Postural	72	55	67	79	62	73	75	60	70
Underweight	72	75	73	78	73	77	76	74	75

Defects of Posture.—Comparing the results of the examinations forming the basis of this study with those made in large numbers in New York and Philadelphia indicates that much remains to be accomplished in working out and using more effective methods of diagnosis

of postural defects. In the latter city fewer cases of flat foot, for instance, were recorded as a result of nearly 150,000 examinations than are here found in 100.

TABLE 13.—Distribution of Postural Defects

Defects	Little Wanderers' Home			Massachusetts General Hospital		
	Male, per Cent.	Female, per Cent.	Both Sexes, per Cent.	Male, per Cent.	Female, per Cent.	Both Sexes, per Cent.
Lateral curvature	15	15	15	13.5	28	22
Round shoulders	43	39	42	66	65	65
Flat foot	9	0	6	8.5	9	9
Bow legs	0	0	0	0	1	0.5
Rickets	7	8.5	6.8	1	0	0.5
Enteroptosis	0	3.5	1.2	0	0	0
Defects	74	66	71	89	103	97
Cases	52	45	50	67	73	70

The incidence of other defects among cases having postural defects shows a positive correlation in every sex division but one. The excess runs from 10 to 23 per cent. among the members of the Little Wanderers' Home section, and from 16 to 23 per cent. in the Massachusetts General Hospital figures. In three out of four divisions the nasopharyngeal obstructions show 9 and 10 per cent. greater incidence, and underweight appears more definitely than it does in the other correlations studied, amounting to 16 and 4 per cent. among boys and girls of the Little Wanderers' Home section, and 4 and 23 per cent. among the others.

TABLE 14.—Incidence of Nasopharyngeal, Teeth and Underweight Defects in Postural Defect Group

Defects	Postural Defects								
	Male			Female			Both Sexes		
	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.
Little Wanderers' Home									
Nasopharyngeal	91	81	86	90	81	85	90	81	85
Teeth	64	54	59	69	46	56	65	51	58
Underweight	34	18	26	29	25	27	32	21	27
Mass. General Hospital									
Nasopharyngeal	98	98	98	97	88	95	98	98	96
Teeth	76	60	71	75	62	71	75	61	71
Underweight	74	70	73	83	60	77	79	65	75

In the tables dealing with the various forms of postural defect, those for flat foot and lateral curvature show each a correlation with round shoulders. The former also shows a greater incidence of lateral curvature, while lateral curvature indicates in three out of the four sex divisions a greater frequency of teeth defects.

When we turn to the figures on round shoulders, we find positive correlations in all but one of the sixteen divisions. This is most marked in relation to "other postural defects"; second, in relation to teeth; third, to underweight, and least, to nasopharyngeal obstruction.

TABLE 15. — INCIDENCE OF VARIOUS OTHER DEFECTS IN ROUND SHOULDERS, LATERAL CURVATURE, AND FLAT FOOT SUBGROUPS

Defects	Male			Female			Both Sexes		
	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.
Round Shoulders									
Little Wanderers' Home									
Other postural	31	15	22	39	10	21	33	13	21
Nasopharyngeal	91	84	87	88	83	85	90	83	86
Teeth	66	53	59	70	47	56	67	51	58
Underweight	35	20	26	30	25	27	33	21	26
Mass. General Hospital									
Other postural	30	9	22	39	10	21	33	13	21
Nasopharyngeal	98	98	98	88	83	85	90	83	86
Teeth	80	64	75	70	47	56	67	51	58
Underweight	77	66	73	85	60	77	81	63	75
Lateral Curvature									
Little Wanderers' Home									
Round shoulders	63	13	22	83	30	38	70	19	27
Nasopharyngeal	97	85	87	75	87	75	90	85	87
Teeth	52	60	59	83	51	56	62	57	59
Underweight	26	26	26	58	22	27	36	24	26
Mass. General Hospital									
Round shoulders	85	47	53	87	59	68	80	53	61
Nasopharyngeal	100	98	98	100	92	95	100	95	97
Teeth	77	70	71	75	70	71	75	70	71
Underweight	69	73	72	77	77	77	75	76	75
Flat Foot									
Little Wanderers' Home									
Round shoulders	57	41	43	..	..	..	..	..	..
Lateral curvature	35	15	17	..	..	..	..	..	..
Teeth	50	60	59	..	..	..	..	..	..
Nasopharyngeal	93	87	87	..	..	..	..	..	..
Underweight	28	26	26	..	..	..	..	..	..
Mass. General Hospital									
Round shoulders	85	62	64	24	66	62	50	65	63
Lateral curvature	23	15	15	48	29	31	37	23	24
Teeth	54	72	93	95	70	71	77	71	71
Nasopharyngeal	92	99	98	95	96	95	94	97	97
Underweight	61	74	73	71	78	77	67	76	75

Underweight Group.—The incidence of other defects among those falling from 7 to 10 per cent. underweight for their height is most marked in the case of round shoulders and the general posture group. This is shown in Table 16.

From this table it is evident that the incidence of persons suffering from nasopharyngeal obstruction is not marked among those who are underweight, but a study of the actual number of defects of this type shows a greater correlation in this respect as well as in the average number of defects of all kinds for each individual. In both instances, the correlation is positive in all four divisions.

When, however, account is taken of the actual number of carious teeth reported, the incidence is reversed. These relationships are shown in Table 17.

TABLE 16.—INCIDENCE OF NASOPHARYNGEAL, TEETH, POSTURAL, ROUND SHOULDERS, LATERAL CURVATURE AND FLAT FOOT DEFECTS IN UNDERWEIGHT GROUP

Defects	Underweight								
	Male			Female			Both Sexes		
	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.	Cases Showing, per Cent.	Cases Not Showing, per Cent.	Total Cases, per Cent.
Little Wanderers' Home									
Nasopharyngeal	86	87	87	83	86	85	85	87	86
Teeth	55	60	59	48	60	56	52	60	58
Postural	67	46	52	48	43	44	60	45	49
Round shoulders	56	38	43	44	37	38	52	37	41
Lateral curvature	17	17	17	30	80	14	22	14	16
Flat foot	10	8	9	..	..	..	..	..	..
Mass. General Hospital									
Nasopharyngeal	98	98	98	95	95	95	96	97	97
Teeth	70	73	71	72	67	71	72	70	71
Postural	69	64	67	79	53	73	75	60	71
Round shoulders	68	55	64	75	45	68	72	50	66
Lateral curvature	15	18	16	31	31	31	24	25	24
Flat foot	7	11	8	8	11	9	8	11	9

TABLE 17.—INCIDENCE OF TOTAL NUMBER OF DEFECTS OF ALL KINDS, ALSO NASOPHARYNGEAL AND TEETH DEFECTS IN UNDERWEIGHT GROUP

	Little Wanderers' Home				Massachusetts General Hospital			
	Male		Female		Male		Female	
	Underweight		Underweight		Underweight		Underweight	
	Cases Showing	Cases Not Showing	Cases Showing	Cases Not Showing	Cases Showing	Cases Not Showing	Cases Showing	Cases Not Showing
Average number of all physical defects	5.6	5.1	6.2	5.0	6.9	6.5	6.7	6.2
Average number of nasopharyngeal defects	2.7	2.6	3.3	3.0	3.8	3.4	3.4	3.3
Average number of carious teeth	3.3	4.0	4.7	4.0	4.1	4.4	3.6	4.0

III. RELATION TO MENTAL RETARDATION

At the Little Wanderers' Home mental examinations were also made.[3] Modified Binet-Simon and Yerkes-Bridges scales were used. The complete records of 155 cases have been placed in three groups: (1) those who received a grade of more than one year above normal expectation; (2) those less than a year above or below normal; (3) those a year or more below normal. The differences are not sufficiently large to make an evident case, but their general trend is suggestive.

3. By Rose S. Hardwick, A.M.

The correlation between degree of mental retardation and total number of physical defects, nasopharyngeal defects, and carious teeth is striking. On the other hand, the worst postural conditions appear to be in the superior mental group, and the worst conditions of underweight in the medium mental group.

TABLE 18.—Summary Showing Relative Incidence Between Various Groups and Subgroups

In making the following list the amounts of the total incidence, both positive and negative, in the four sex divisions have been taken as a rough index of the degree of incidence shown among the various groups studied. The pairs of terms are arranged in the order which the totals indicate.

1. Lateral curvature and round shoulders
2. Nasopharyngeal and teeth (see 12)
3. Round shoulders and other postural defects
4. Round shoulders and teeth
5. Underweight and round shoulders (see 9a)
6. Teeth and posture (see 7)
7. Posture and teeth (see 6)
8. Underweight and posture (see 10)
9a. Round shoulders and underweight (see 5)
9b. Nasopharyngeal and posture (see 13)
10. Posture and underweight (see 8)
11a. Lateral curvature and underweight
11b. Lateral curvature and teeth
12. Teeth and nasopharyngeal (see 2)
13. Posture and nasopharyngeal (see 9b)

TABLE 19.—Incidence of Physical Defects of All Kinds, Also Nasopharyngeal Defects, in Three Mental Grades

	(1) 1 Year or More Above Scale	(2) Less than 1 Year Above or Below Scale	(3) 1 Year or More Below Scale
Average number of physical defects of all kinds	4.6	4.8	5.1
Average number of nasopharyngeal defects	1.9	2.2	2.4
Per cent. defects of all kinds	15	33	52
Per cent. nasopharyngeal defects	74	84	91
Per cent. teeth defects	39	57	63
Per cent. postural defects	61	47	55
Per cent. underweight	17	37	28

SUMMARY

The evidence presented points to the following general statements:

1. Children reported to be sufficiently well to attend school, and to engage in the ordinary activities of normal children, were found to average 5.2 physical defects of all kinds and 2.5 nasopharyngeal defects (Table 1).

2. Children brought to a hospital clinic for examination and treatment showed an average of 6.8 defects of all kinds and 3.5 nasopharyngeal defects (Table 1).

3. The distribution of number of defects according to age was remarkably uniform. In each group studied the largest number of children were between the ages of seven and nine. These years showed the greatest average number of defects in the Massachusetts General

Hospital group (7.2), but in the Little Wanderers' Home group the highest average number of defects (6.0) was found in the years from ten to twelve. There is no evidence of any particular age at which children have an especially large number of defects (Table 2).

4. Only nine children of the six hundred and two examined were found to be free from defects (Table 3).

5. The nasopharyngeal defects appeared in a larger number of cases (Little Wanderers' Home, 87 per cent., Massachusetts General Hospital, 96 per cent.), and also totaled a greater number of defects than any other group (Little Wanderers' Home, 49 per cent. of all defects found, Massachusetts General Hospital 53 per cent. of all) (Table 3).

6. The most striking differences in percentages of physical defects found in the comparison with results secured in public schools in New York and Philadelphia are in hearing, nasopharyngeal obstruction, malnutrition and posture (Table 5).

7. In one of our largest and best conducted children's clinics about half the defects found in the examinations were brought down in the summary for diagnosis purposes. The complete examination of the same children made in our nutrition clinic showed that the general examination had found only 44 per cent., and the diagnosis summary had recorded less than 21 per cent. of the defects revealed in the nutrition examination. The greatest discrepancies occurred in nasopharyngeal, postural and teeth defects, in the order named (Table 8).

8. The correlation between the total number of nasopharyngeal defects and those of underweight was much more clearly marked than that between the number of nasopharyngeal cases and underweight (Table 17).

9. The correlation between the actual number of carious teeth found and underweight was negative in three out of four of the sex groups (Table 17).

10. The summary of relative incidence indicates that postural defects, especially round shoulders, have greater value for diagnosis purposes in malnutrition than is usually assigned them (Table 18).

11. The mental tests made in the Little Wanderers' Home group showed positive correlation between mental retardation and the total number of all physical defects, nasopharyngeal, and teeth defects (Table 19).

I wish to express my appreciation of the cooperation in this work of the Children's Department at the Massachusetts General Hospital, and of the personal assistance of Dr. Charles L. MacGray in physical examinations; of Dr. Charles B. Porter, Dr. George Smith, Dr. D. Crosby Greene, and Dr. George Tobey in nose and throat examinations; of Dr. Francis Rackemann and Dr. J. P. Turnbull in proteid tests; of Dr. C. Morton Smith on hereditary syphilis; and of Dr. George Holmes in roentgen-ray work.

THE INFANT OF LOW BIRTH WEIGHT; ITS GROWTH AND DEVELOPMENT*

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The "impulse to grow" is being recognized as a very interesting and important phenomenon in the study of infancy and childhood. The growth in utero seems to be divided into two well defined parts; that of the first six months, and that of the last three months. Of the first period, not so much is known, for the opportunities to study this growth are relatively few. The second period, that of the last three months in utero, has been rendered very important from the pediatric point of view, firstly, because there is great growth during this period, and secondly, because this period is unfortunately often spent outside, instead of inside the uterus. It has always been an interesting theme to know whether:

(a) if the infant, although of full term, for some unknown reason does not obtain this added growth of the last three months and comes into the world very much underweight and underdeveloped, or,

(b) if the infant is born prematurely and thus fails to obtain this stimulus of the last few months, does it develop more rapidly than the normal (after being born), or, does it lag behind physically and mentally, and when, if ever, does it catch up to the normal?

It was in the endeavor to answer some of these questions that we have followed our cases. This opportunity was given us under the supervision of the John E. Berwind Maternity Clinic, a free outdoor maternity clinic. These children were delivered in their homes. Soon after birth a nurse with pediatric experience visited them, weighed and cared for them, repeated her visits daily for three days, then again visited them on the seventh and fourteenth days, urged breast feeding and instructed the mother. When 4 to 6 weeks old, the children were brought to the clinic and thereafter seen monthly or more often, if necessary, for one year or more.

We have had 272 children with low birth weights in 11,100 deliveries, an incidence of 25 per 1,000 pregnancies (2.5 per cent.). We have arbitrarily made 2,500 gm. or under, the weight designated as low birth weight.

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Miller [1] collected 6,000 cases of low birth weight in 121,626 deliveries, an incidence of 5 per cent. Ylppo [2] had 114 cases in 2,168 deliveries, or 5 per cent. and Schmitt [3] had 316 cases in 8,516 deliveries, or 3.6 per cent. Holt and Babbitt [4] had 183 cases out of 9,160 deliveries with a birth weight under 5 pounds, or about 2 per cent. It would appear, therefore, that from 2 to 5 per cent. of all pregnancies result in infants (a) that have either been born prematurely, (b) are one of twins and have had their growth retarded at the expense of the other twin, or (c) a full term infant of low birth weight.

The material as shown in Table 1 consisted of 163 cases from single pregnancies, and 109 from twin pregnancies. The twin pregnancies at times represent both children from the one mother; at other times, one twin, the other being an infant with a normal weight. The infants were deemed premature, first by means of the history, as far as possible, and by weight, length and other clinical signs.

Of the 272 infants, three were under 1,000 gm. weight, sixteen were between 1,000 and 1,500 gm., forty-nine were between 1,500 and 2,000 gm. and 204 were between 2,000 and 2,400 gm. weight.

TABLE 1.—Analysis of Cases

272 live born children; 163 single pregnancies; 109 twin pregnancies

	Number Under 1,000 Gm.	No. from 1,000 to 1,500 Gm.	No. from 1,500 to 2,000 Gm.	No. from 2,000 to 2,400 Gm.	Total
Single pregnancies.....	1	7	21	134	163
Twin pregnancies.....	2	9	28	70	109
Total number.....	3	16	49	204	272

Of these cases, 166 were followed for three months, 130 for six months, 100 for twelve months and twenty-six a greater length of time. We lost by death and otherwise almost one-half of the cases before the end of the first three months.

MORTALITY

It is difficult to obtain the mortality rate of these children with any degree of accuracy because of the inabilty to keep track of this class of population over long periods of time. It seemed more advisable to

1. Miller, N. Th.: Jahrb. f. Kinderheilk. **25**:179, 1886.
2. Ylppo, A.: Ztschr. f. Kinderheilk. **24**:1, 1919.
3. Schmitt, H.: Ztschr. f. Geburtsh. u. Gynäk. **81**:382, 1919.
4. Holt, L. E., and Babbit, E. L.: J. A. M. A. **64**:287 (Jan. 23) 1915.

give the mortality figures of the first day, the first month and approximately for the first year. The early infant mortality of these children is absolutely accurate for very few have been lost sight of during the first month. From Table 2 it will be seen that the mortality rate for the first twenty-four hours lies between 9 and 13 per cent., or from 90 to 130 per 1,000 born; for the first week the rate lies between 14 and 20 per cent., or from 150 to 200 per 1,000 born.

TABLE 2.—MORTALITY DURING THE FIRST TWENTY-FOUR HOURS

Author	Cases	Deaths	Mortality, per Cent.
Ylppo	668	62	9.0
Schmitt	316	44	13.9
Schwarz and Kohn	272	29	10.5

TABLE 3.—MORTALITY DURING THE FIRST WEEK

Author	Cases	Deaths	Mortality, per Cent.
Ylppo	668	133	20
Schwarz and Kohn	272	39	14

The mortality figures for the first month are not so accurate. Tables 4 and 5 have been prepared from our own material and also from figures taken from the literature. For more accurate orientation we have grouped all the cases (Table 5) according to birth weight. Under 1,000 gm. the mortality rate is 940 per 1,000 born; from 1,000 to 1,500 gm. the mortality rate is 690 per 1,000 born; from 1,500 to 2,000 gm. it is 410 per 1,000 born, and from 2,000 to 2,500 gm. it is 110 per 1,000 born.

Summing up the mortality rate of the first month of life, it will be seen that 130 per 1,000 born, die during first day. From 150 to 200 die during the first week, and about 300 during the first month. Of 4,500 live born children from the same clinic [5] including those of normal and subnormal birth weight, there was a mortality rate of 10 per 1,000 for the first day, 15 per 1,000 for the first week and 26 per 1,000 for the first month. The rate is slightly better than the figures quoted from hospitals where undoubtedly more difficult cases are delivered and thus explain the slight increased rate; for instance, reports of Holt and Babbitt in New York and Kerness [6] in Munich, their mortality rate being 2.5 per cent. for the first week.

5. Schwarz, H.: Tr. Am. Soc. Prevention of Infant Mortality, 1914, p. 167.
6. Kerness, S.: Ztschr. f. Kinderheilk. Ref. **4**:19, 1913.

TABLE 4.—MORTALITY OF THE FIRST MONTH IN RELATION TO BIRTH WEIGHT

Author	No. under 1,000 Gm.	Mortality, per Cent.	No. from 1,000 to 1,500 Gm.	Mortality, per Cent.	No. from 1,500 to 2,000 Gm.	Mortality, per Cent.	No. from 2,000 to 2,500 Gm.	Mortality, per Cent.	Total Number	Total Mortality, per Cent.
Crede[7]	...	..	24	83	15	36	54	11	93	31
Bakker[8]	9	100	75	88	138	34	502	10.5	724	16
Maygrier[9]	...	..	46	68	141	34	548	6	735	13
Schmitt[3]	15	100	48	80	113	41	83	11	273	13
Detre[10]	...	..	...	..	6	66.6	53	16	59	21
Ladd[11]	...	..	32	71.9	50	40	...	..	125	65
Budin, 1898-99 [12]	24	96	92	92	293	68.6	124	37	433	80
Yippo[2]	37	83	183	48	240	23	208	14.9	668	30
Francois[13]	9	89	31	62	66	27	310	7	416	18.7
Schwarz	3	66	16	87.9	49	31	204	14	272	20

TABLE 5.—MORTALITY OF FIRST MONTH

Weight	Total Number Cases Selected from Literature	Total Number Deaths	Mortality, per Cent.	Mortality per 1,000 Pregnancies
Under 1,000 gm.	97	91	94	940
1,000 to 1,500 gm.	547	371	69	690
1,500 to 2,000 gm.	1,111	457	41	410
2,000 to 2,500 gm.	2,086	232	11	110
Total	3,841	1,151	30	300

TABLE 6.—MORTALITY FOR THE FIRST YEAR

Weight	Alive	Dead	Lost	Total Cases	Per Cent. Mortality of Known Cases
Under 1,000 gm.	1	2	0	3	66
1,000 to 1,500 gm.	3	10	3	16	77
1,500 to 2,000 gm.	11	22	16	49	50
2,000 to 2,400 gm.	84	47	73	204	36
Total	99	81	92	272	45

Table 6 shows the mortality for one year. Inasmuch as it was impossible to know the fate of all of our cases, we took the number known to be alive and those known to be dead and computed the mortality rate from these combined figures. Most likely, about the same percentage of mortality affected those lost to us. This table shows that 450 per 1,000 born, die before the end of the first year as compared with the general infant mortality rate of New York City of 90 per 1,000 born.

7. Crede, B.: Arch. f. Gynäk. **24**:128, 1884.
8. Bakker, J.: Mitth. a. d. Hamburger Staats Krankenanstalt, 14 (Sept.) 1913.
9. Maygrier, C.: Obsteterique **12**:290, 1902.
10. Detre, G. R.: L'avenir des prematures (etc.), Thésé de Paris, 1912.
11. Ladd, M.: Arch. Pediat. **27**:416, 1910.
12. Budin, P.: Obstetrique **4**:107, 1899.
13. Francois, M.: Caractére et elevage des prematures, Thésé de Paris, 1903.

MORTALITY IN RELATION TO PREMATURITY

From Table 7 it will be seen that prematurity is a most important factor in causing the high mortality in these cases. The premature cases have a mortality of 425 per 1,000 born as compared with 233 per 1,000 born in the full term cases. Single pregnancies premature, as compared with single pregnancies full term, show a mortality of 410 in the former to 157 in the latter, showing that prematurity is an important factor.

TABLE 7.—MORTALITY IN RELATION TO PREMATURITY

	Total Number Cases	Total Number Deaths	Mortality, per Cent.	Deaths per 1,000 Pregnancies
Single and Twin Pregnancies:				
Prematures	158	68	42.5	425
Full term	114	27	23.3	233
Single Pregnancies:				
Premature	89	37	41.0	410
Full term	69	11	15.7	157
Twin Pregnancies:				
Premature	69	31	44.3	443
Full term	45	16	34.8	348
Premature Pregnancies:				
Single	89	37	41.0	415
Twin	69	31	44.3	443
Full Term Pregnancies:				
Single	69	11	15.7	157
Twin	45	16	34.8	348

Comparing premature twin pregnancies to full term twin pregnancies, there is a mortality of 443 per 1,000 and 348 per 1,000 in the latter. Here prematurity still shows its baneful effect, but not so markedly.

Discussing prematurity as to its effect on single pregnancies as compared with twin pregnancies, it will be seen that the mortality in the former (single) is 410 as compared with 443—not such a great difference—emphasizing that prematurity is more of a factor than twinning.

Finally, comparing full term pregnancies, that is single and twin, there is a mortality of 157 per 1,000 in the former and 348 per 1,000 in the latter (twin), showing that in these children twinning is not as deleterious as prematurity, yet gives only one-third the chance to life.

Of the many standards by which growth and development may be measured, gain in weight is probably the most reliable. Table 8 is the weight chart of eighteen cases with a birth weight under 2,000 gm., followed and weighed at different intervals during the entire first year. It is also interesting from the circumstance that some of the cases are from a social stratum in which all possible safeguards as to life and the best environment were obtained. The average birth weight of all the cases was 1,660 gm. The same children weighed 6,070 gm. at 6 months. This is a gain of 3,360 gm. for the first six months or a

gain of 764 gm. per month. This is a fair, or even a satisfactory gain for any infant at this period of life. At the end of the first year these children had an average weight of 6.690 gm., a gain of 1,920 gm. during the second six months of the first year, or 360 gm. per month, which is, perhaps, slightly below the normal monthly gain. Comparing the two social strata, it will be seen that those with the better niveau gained 1,440 gm. more during the first six months, and about 2,000 gm. more for the entire year. Yet neither one or the other made up for its deficiency at birth. The average gain for the last two months in utero, according to Friedenthal,[14] is something more than 1,920 gm., whereas these children gained 3,300 gm. or 3.25 kg., in the first six months of their extra-uterine life, and about 2,000 of these during the first two months. Thus the impulse seems to get weaker and weaker as these children get older, and it is a common experience that their weight chart becomes flatter after the first six months and, as we will show, still flatter from the twelfth and the eighteenth month. Most of these children were premature; thus the marked impulse to grow, characteristic of the last three months in utero, did not seem to persist.

WEIGHT

TABLE 8.—Weight Chart of Cases Under 2,000 Gm. Birth Weight

	Birth Weight, Gm.	Weight at 6 Mos., Gm.	Weight at 12 Mos., Gm.
Private material.....	1,440	6,480	8,940
	1,440	.....	6,100
	1,590	5,280	8,640
	1,680	5,340	7,680
	1,680	6,660	8,700
	1,860	6,520	8,240
	1,920	5,760	7,440
Clinic material.....	900	4,260	6,090
	1,320	.....	5,280
	1,440	.....	5,340
	1,500	3,840	5,790
	1,680	6,510	7,770
	1,800	4,800	7,920
	1,830	3,480	3,930
	1,960	2,760	3,930
	1,920	4,200	5,820
	1,920	6,600	7,560
	1,920	.....	8,820
Average all cases.....	1,660	5,040	6,990
Average private cases.....	1,650	5,850	8,520
Average clinic cases.....	1,661	4,410	6,540

We have observations on the weight of forty-nine cases with birth weight between 2,000 and 2,500 gm.; seventeen of these were of twin pregnancy and thirty-two of single pregnancy. The average weight

14. Friedenthal, H.: Ergeb. d. inn. med. u. Kinderheilk. **9**:505, 1912.

at six months was 5,800 gm. with a difference of only 180 gm. in favor of the twin pregnancy. At twelve months the average weight was about 8,000 gm. (16 pounds, 9 ounces), showing no difference whatever between the single and twin pregnancy. It would seem that the impulse to grow does not suffer because of twinning. These children gained about 3,500 gm. the first six months, which is about normal, and 2,200 gm. the second six months, which is within the normal. These children then, just as in the case of the still smaller ones, gain about what a child with a normal birth weight would, but do not exceed this and therefore do not catch up.

TABLE 9.—WEIGHT CHART OF SINGLE PREGNANCIES WITH BIRTH WEIGHT OF FROM 2,000 TO 2,400 GM.

6 Months Gm.	1 Year Gm.	6 Months Gm.	1 Year Gm.	6 Months Gm.	1 Year Gm.
4,320	5,550	.....	7,740	.....	8,250
4,320	5,940	6,150	7,810	5,280	8,280
.....	6,240	.....	7,920	6,450	8,280
3,930	6,480	4,800	7,920	.....	8,880
4,980	6,540	5,160	8,070	7,200	8,940
5,490	6,810	.....	8,100	7,620	9,360
4,890	6,960	.....	8,160	.....	9,480
5,730	7,110	.....	8,160	6,480	9.540
5,910	7,200	6,480	8,220	7,170	9,540
.....	7,530	6,960	8,250	7,200	9,600
5,760	7,530				

Total number of cases, 32; average at 6 months, 5,760 gm.; average at 12 months, 8,010 gm.

TABLE 10.—WEIGHT CHART OF TWIN PREGNANCIES WITH BIRTH WEIGHT OF FROM 2,000 TO 2,400 GM.

6 Months Gm.	1 Year Gm.	6 Months Gm.	1 Year Gm.	6 Months Gm.	1 Year Gm.
4,620	5,520	6,480	7,950	.....	8,460
5,160	6,960	.....	8,080	5,100	8,580
.....	6,600	6,060	8,220	6,630	8,760
.....	7,230	5,760	8,340	6,660	8,880
6,480	7,560	.....	8,370	.....	9,000
.....	7,700	6,120	8,430		

Total number of cases, 17; average at 6 months, 5,940 gm.; average at 12 months, 7,920 gm.

LENGTH

The growth in length is, perhaps, next in importance to the weight, yet hereditary factors probably play a greater rôle here. In the cases with a birth weight under 2,000 gm. (Table 11) none attained a height as great as the normal infant. The length at one year was never more than 64 cm., the average length of a five months infant. The weight at birth in the five cases observed seemed to have some relation to the length at one year, for the 990 gm. infant was the smallest in length. Although it was the smallest in length at the end of the year, it was as heavy as those with a length of 64 cm. The rate of growth during

the second six months in two cases was from 7 to 8 cm., equal to that of normal children.

TABLE 11.—Length Chart of Twin and Single Pregnancies with Birth Weight Below 2,000 Gm.

Birth Weight, Gm.	Length at 1 Month, Cm.	Length at 6 Months, Cm.	Length at 1 Year, Cm.	Weight at 1 Year, Gm.
990	34.4	53	60.9	6,150
1,440	..	..	64	5,340
1,800	..	..	64	5,340
1,920	49	55	63	6,810
1,920	..	..	64	.....

TABLE 12.—Length Chart of Single Pregnancies with Birth Weight Between 2,000 and 2,400 Gm.

Length at 6 Months, Cm.	Length at 1 Year, Cm.	Weight, Gm.	Length at 6 Months, Cm.	Length at 1 Year, Cm.	Weight, Gm.
..	60	5,550	..	69	9,390
..	63	.....	..	70	7,920
54	64	7,800	..	70	8,880
..	64	6,180	61	70	8,280
60	65	7,110	..	70	8,280
57	66	7,920	..	70	7,890
..	66	5,940	..	71	8,160
60.5	66.5	7,230	68	71.7	6,960
..	66.7	7,740	60	72	9,540
..	67	8,220	63	72	9,690
..	67	8,070	..	72	9,690
..	67	7,530	66.5	73	9,480
62	67.5	8,250	..	72	9,360
60	68.3	7.200			

Total numoer of cases, 28; average length at 6 months (11 cases), 61 cm.; average height at 12 months, 68 cm.

TABLE 13.—Length Chart of Twin Pregnancies with Birth Weight Between 2,000 and 2,400 Gm.

Length at 6 Months, Cm.	Length at 1 Year, Cm.	Weight, Gm.	Length at 6 Months, Cm.	Length at 1 Year, Cm.	Weight, Gm.
57	62	6,880	..	68	8,210
60	65	7,560	61	68.5	9,060
..	66	5,520	57	68.9	.....
..	67	8,370	61	69	8,880
..	67	7,200	..	70	8,460
60	67.6	7,950			

Total number of cases, 11; average length at 6 months, 59.3 cm.; average length at 1 year, 67.3 cm.

In Tables 12 and 13 we have collected thirty-nine cases with a birth weight of between 2,000 and 2,400 gm., of which twenty-eight are from single pregnancies and eleven from twin pregnancies. At six months, seventeen of these thirty-nine cases have an average length of 60 cm.; about four are below the average of our general clinic material, and at one year showed an average length of 67.5 cm., as compared with an average of 73 cm. for the same type of child. It

appears that the growth in length lags about the same as the growth in weight during the entire first year. The growth in length of twins compared with those of single pregnancies shows the same phenomena as the weight figures, there being very little difference at any time. The impulse to grow in length for the first year is not strong enough to overcome the handicap at birth.

FURTHER DEVELOPMENT

The development of these children beyond the first year is naturally very interesting and important. Up to this period these cases do not make up what they had been deprived of at birth. Very few have

TABLE 14.—WEIGHT AND DEVELOPMENT OF CHILDREN WITH LOW BIRTH WEIGHT AFTER THE FIRST YEAR

Birth Weight	Age When Last Seen	Weight at One Year	Weight When Last Seen	Average Gain per Year after First Year	Length When Last Seen	Development and Remarks
2,200	15 mos.	.....	9,030	.....	...	General condition poor
2,200	15 mos.	.....	.....	.....	...	Died of pneumonia
1,440	15 mos.	.....	.....	.....	...	Died of pneumonia
1,680	17 mos.	8,700	10,680	3,900	...	Fair
1,860	17 mos.	8,220	9,660	2,800	...	Fair
1,680	18 mos.	7,680	9,420	3,480	...	Fair
1,830	18 mos.	3,960	7,770	7,660	...	Poor
2,300	2 yrs.	.....	11,580	.....	...	Good
1,440	2 yrs.	5,100	7,920	2,820	...	Poor
2,120	2 yrs.	9,120	11,640	2,520	...	Good
2,260	2 yrs.	.....	10,260	.....	...	Fair
2,400	2 yrs.	9,120	10,320	1,200	...	Fair
2,280	2 yrs.	.....	.....	.....	...	Died of tuberculosis
1,920	2 yrs.	7,380	1,010	6,370	...	General condition fair
2,400	2 yrs.	.....	.....	.....	...	General condition excellent
1,440	2 yrs.	8,940	10,740	1,800	81	General condition good
2,160	2 yrs.	.....	.....	.....	...	General condition poor
2,160	2 yrs.	.....	.....	.....	...	General condition excellent
2,160	2 yrs.	9,120	9,210	90	...	General condition fair
2,280	2 yrs.	.....	8,700	.....	70	General condition good
1,920	2 yrs.	8,820	8,580	"—"240	...	General condition poor
2,400	2 yrs.	.....	9,720	.....	73	General condition fair; 4 teeth
2,160	2 yrs.	.....	.....	.....	...	General condition good
2,160	2½ yrs.	.....	13,200	.....	86	General condition excellent
1,500	2½ yrs.	5,760	9,360	400	76	General condition fair
1,800	3 yrs.	9,600	14,400	2,400	...	General condition fair
2,400	3 yrs.	.....	.....	.....	...	Does not talk, walked at 2 yr.
1,320	3½ yrs.	7,380	10,140	740	83	General condition poor; Hb. 28 per cent.
2,400	3½ yrs.	.....	.....	.....	...	Stupid; does not walk
2,400	4 yrs.	7,740	13,080	1,780	89	Excellent
2,040	4 yrs.	6,360	10,560	1,400	91	Fair
1,680	4 yrs.	.....	.....	.....	...	General condition good
1,920	5 yrs.	9,240	27,480	4,560	...	General condition good
2,400	5 yrs.	.....	.....	.....	...	General condition good
2,400	6 yrs.	.....	19,680	.....	...	Excellent
2,160	6 yrs.	.....	.....	.....	...	Excellent
2,400	6 yrs.	.....	.....	.....	...	Excellent
990	6 yrs.	5,880	21,600	3,144	117	Good
2,160	10 yrs.	6,900	19,140	1,360	114	Good

attained an approximately normal weight or length. Table 14 shows the follow-up of thirty-nine cases. Seven cases were followed for from fifteen to eighteen months; eighteen, from two to three years; seven, from three to four years; six, from five to six years, and one was followed for ten years.

Of the seven cases followed from fifteen to eighteen months, two had died of pneumonia; only one infant had reached an approximately normal weight (10.68 kg.); two were in very poor general condition, and three were rated as fair. The weights of these ranged from 7 to 9 kg. (15 to 19 pounds). Of the eighteen cases followed from two to three years, one died of tuberculosis; three were in poor condition; six were rated fair, and eight, about 50 per cent., were in good physical condition. We have been able to secure the weights of thirteen of these cases. Only three of them can be called normal (11.6, 11.6 and 13.0); five weighed from 7 to 9 kg. and from 9.6 to 10.5 kg., all decidedly under weight. Thus of the preceding twenty-five cases only four had reached their normal weight in or before the fourth year. We have the growth in length in five cases, two measuring 81 and 86 cm., well within the average for this age; the remaining three were between 70 and 76 cm., distinctly below the normal of a 2 to 3 year old child.

We have followed nine cases from four up to six years. Two of these are mentally subnormal; one is in very poor physical condition; two are rated fair, and four are in good physical condition.

The weight in five of these cases was obtainable and found to be normal in only one case (27.5 kg.); the others weighed 14.4, 10.1, 13.4 and 10.5 kg., respectively. We have the lengths of three of these children. They ranged from 83 to 91 cm. for 3½ and 4 year old children, which is, perhaps, not quite up to normal, but nearly so. We have records of five cases above 6 years, all in good condition. The weights are normal, except in the 10 year old child, whose stature is very small (114 cm.), but whose general condition is good.

The column in Table 14 showing average gain per year is extremely interesting, for it will be seen that in most cases the average gain is above the normal. As shown before, this is not true of the first twelve months where the gain is either about the average, normal or less. It would seem, therefore, that these children gain slightly above normal the first six months. After the first year these children gradually make up the deficiency in weight, but rarely do they attain the normal weight before the end of the fourth year (5 in 25). This is in accord with the finding of Ylppo.[2] The gain in weight does not keep pace with the gain in length however, for although we have not enough data for a positive statement, this deficiency in length is made up before that of the weight.

SUMMARY

From 2 to 5 per cent. of all viable births result in children of low birth weight.

The mortality rate during the first month in this type of case is ten times that of the normal.

The lower the birth weight, the greater the mortality.

The mortality rate for the year in this type of case is four and one-half times the normal.

In these children of low birth weight the mortality is twice as great in the premature as in the full term.

Twinning in the premature does not markedly affect the mortality rate.

In the full term, the single pregnancy has twice the chance for life as compared with the twin pregnancies.

The gain in weight during the first twelve months is at the same rate as that of the normal child, so that the deficiency is not made up at the end of the first year. Twins do not act differently than those from single pregnancies.

The growth in length is not made up during the first twelve months.

These children seem to attain the normal in length sooner than the normal in weight. It is only before the end of the fourth year that they compare with children of normal birth weight.

The general condition and mentality seem to be that of normal children through infancy and early childhood, although they have a greater tendency to anemia and rickets.

EFFECT OF A RATION LOW IN FAT SOLUBLE "A" ON THE TISSUES OF RATS*

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It is an established fact that the fat soluble vitamin is necessary in the ration. Drummond [1] reported a lowered resistance to infection in rats deprived of this vitamin. He found, however, no specific pathologic change on gross examination of grown or half grown rats. Emmett [2] states also that in a histologic examination of rats fed a ration deficient in the fat soluble vitamin he found the tissues normal. He does not give the age of the animals.

On the other hand, Tozer [3] found that young guinea-pigs on a ration deficient in the fat soluble vitamin, even with an excess of the antiscorbutic vitamin, develop enlargements of the costochondral junctions very similar to the beadings of scurvy.

M. Mellanby,[4] who worked with puppies, found that the fat soluble vitamin controlled the calcification of the teeth. With a ration of white bread and 200 c.c. skimmed milk daily, the shedding of the deciduous teeth was retarded, the development of the permanent dentition was delayed, the arrangement of the teeth in the jaws was poor, and the calcium content of the teeth was subnormal. The teeth of the more rapidly growing puppies were worse. If the puppy was over 3 months old when put on the ration, the teeth were not affected to any degree.

E. Mellanby [5] has shown that the fat soluble vitamin is largely responsible for the calcification of bone. With the bread and skimmed milk ration, puppies developed a beading of the ribs. This is more marked in puppies one month old than in those 6 weeks old. After eight weeks they failed to develop the beaded rib. This lesion appears to be outgrown in time, and resembles the condition of rickets in children.

The two lines of investigation which we have followed are: (1) a long time experiment with a ration low in the fat soluble vitamin but satisfactory in other respects; (2) an experiment with young animals on a ration low in the fat soluble vitamin. We are not as yet ready to report the results of the second experiment.

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* Received for publication, Nov. 3, 1920.

* From the Home Economics Laboratory, University of Wisconsin.

1. Drummond: Biochem. J. **13**:95, 1919.
2. Emmett: J. Biol. Chem. (Proceedings) **41**: liii, 1920.
3. Tozer: Quoted by Delf, Biochem. J. **14**:212, 1920.
4. Mellanby, M.: Lancet **2**:767, 1918.
5. Mellanby, E.: Lancet **1**:407, 1918.

It was necessary to choose a ration which was eaten readily, for there are marked pathologic changes in inanition. Jackson [6] found that a limited amount of whole wheat bread and whole milk produced definite changes in the suprarenal of rats. McCarrison [7] reports a profound effect of inanition on the gastro-intestinal tract of monkeys which had refused to eat a diet of autoclaved rice. In fowls he found hypertrophy of the suprarenal with an increased amount of epinephrin. Other organs were greatly atrophied.

TABLE 1.—Comparison of Weights of Organs of Donaldson's and Authors' Experiment Animals

	Age	Body Weight, Gm.	Two Kidneys, Gm.	Spleen, Gm.	Testes, Gm.
Donaldson	...	204.3	1.74	0.545	2.203
No. 15	114	203.0	2.0	1.0*	2.8
Donaldson	...	178.1	1.544	0.479	2.080
No. 15	114	178.0	1.3	0.65	2.5
Donaldson	...	175.0	1.521	0.471	2.007
No. 15	114	174.0	1.6	0.9	1.0†

* Enlarged spleens are common in stock rats.
† Only one testes found.

TABLE 2.—Growth of Second Generation Born on the Ration

Serial No. of Litter	Age at Death, Days	Maximum Weight, Gm.	Normal Weight, for Age, Gm.
26	101	89	165
	60	26	77
	50	32	59
	100	98	165
	70	51	104
	60	40	80
	40	20	43
	75	64	118
	70	42	98
	90	71	150
	50	22	59
27	Discontinued at 25 days	32	25
	Discontinued at 25 days	35	25

Rats were employed because the span of life is shorter and we desired to continue the ration through as many generations as possible. The ration consisted of crushed oats, polished rice and skimmed milk ad libitum, and a limited amount of cooked potato and egg white. On this ration, the half grown males grew at from 80 to 90 per cent. of the normal rate for three months. After a period of maintenance, they became emaciated and died. None lived longer than eight months.

Most of the females reared one litter and a few reared two litters on the ration. All but three of the litters were born in the first two

6. Jackson: Am. J. Anat. **21**:321, 1917.
7. McCarrison: Indian J. M. Res. **7**:269, 1919.

months. A second litter of four young was born when the mother had been 148 days on the ration.

One of the four was a female. She reared a litter of two young. She was 135 days old at parturition, which made 283 days since the beginning of the experiment. When the young were twenty-five days old, they were discontinued. This made 308 consecutive days for the three generations.

Of the twenty-three females born and continued on the ration, seven produced litters. Only two of these litters were reared. There were eleven young in one litter. They were born when the mother was 131 days old and 144 days after the original rats were put on the ration. Two of the eleven lived 101 days, which made a total of 285 days of the experiment.

Distended abdomens were conspicuous in all of the young born on the ration. In some cases this disappeared after four or five months. Xerophthalmia, which Osborne and Mendel [8] and McCollum and Simmonds [9] reported in the absence of the fat soluble vitamin, appeared at from four to five months in the rats born on the ration. In less than 200 days, all of the animals had either died or were chloroformed in an emaciated condition. Three rats born on the ration were killed when they were still growing and in good condition. The testes, kidneys and spleen were weighed, and the weights were compared with those given by Donaldson [10] for rats of the same weight. Table 1 shows that these organs are of about normal weight.

In the litter of eleven young of the second generation born on the ration, in addition to the distended abdomen and xerophthalmia, small hemorrhagic spots appeared on the tails, and when the rats were about 30 days old the ends of the tails dropped off. The eyes were pale and a blood count showed from 4,000,000 to 5,000,000 red blood cells in contrast to a normal count of 8,000,000. Table 2 gives the growth in this generation.

A postmortem examination of the rats put on the ration, as well as of those born on the ration, showed the stomach and intestines filled with gas, the intestines dull and brown, and no intraperitoneal fat visible.

Necropsy of the second generation born on the ration revealed very marked anemia. The lungs were white and the liver and kidneys were sand color. The bones were very fragile. There was no apparent beading of the ribs.

A histologic examination was made on rats from most of the litters. The organs examined were: heart, spleen, liver, lung, pancreas, kidney

8. Osborn and Mendel: J. Biol. Chem. **16**:431, 1913.
9. McCollum and Simonds: J. Biol. Chem. **32**:347, 1917.
10. Donaldson: Memoirs of Wistar Institute of Anat. and Biol., No. 6, 1915.

and testes. The tissues were removed immediately at necropsy and fixed in Zenker's fluid, embedded in paraffin, cleared in xylol, and stained with hematoxylin and eosin. This stain gives only the marked differences. Differential stains were not used.

TABLE 3.—RESULTS OF HISTOLOGIC EXAMINATION OF ORGANS

Lot No.	Animal No.	Days on Ration	Kidney	Spleen	Lung	Heart	Pancreas	Liver
7	3	227	Cloudy swelling	Dilated venous sinuses	*	*	Normal	Normal
8	4	227	Cloudy swelling	Dilated venous sinuses	..	*	Normal	Normal
5	6	217	Slight congestion	Congestion	Congestion	Normal	Normal	Normal
7	8	217	Albuminous precipitation in tubules, slight congestion	*	*	Normal	Normal	Normal
9	9†	93	Slight congestion, albuminous precipitation in tubules	Congestion	Normal	Normal	*	*
15	10†	115	Cloudy swelling	.....	Normal	Slight atrophy of parenchyma	Normal	Normal
	12	115	Cloudy swelling, albumin in tubules	Congestion, dilated venous sinuses	*	*	Normal	..
	14	181	Cloudy swelling, albumin in tubules	Congestion, dilated venous sinuses	*	*	Normal	Normal
18	25†	178	Cloudy swelling, congestion	Extreme congestion	Normal	Normal	Normal	Normal
	26†	178	Cloudy swelling, albumin in tubules	*	*	Normal	Normal	*
	27	178	Cloudy swelling, albumin in tubules	Dilated venous sinuses, slight destruction of tissue	*	Normal	*	*
26	30†	70	Extreme cloudy swelling, cells of tubules skeletonized	Congestion	Bronchopneumonia	Normal	Nuclei indistinct	Absence of red blood cells
	31†	70	Extreme cloudy swelling, cells of tubules skeletonized, glomeruli shrunken	Congestion	Bronchopneumonia	Normal	Normal	Absence of red blood cells

* Not examined.
† Testes in Animals No. 9, 10, 25, 26, 30 and 31 were normal; testes of other animals not examined.

The examination showed that there were very few changes in the tissues on this ration. The heart, pancreas, liver and testes remained normal in most cases. Slight changes were found in the kidney and spleen. Congestion and chronic inflammation was frequently found,

indicating, in all probability, a secondary infection, although an effort was made to discard without microscopic examination all those which gave evidence of infections of the respiratory tract.

Liver: The liver appeared to be normal in all cases. Necrotic and degenerative changes were absent. In Lot 26 there was a total absence of red blood cells. Otherwise the structure was normal.

Spleen: The spleen showed very slight changes. There was congestion with frequent dilation of venous sinuses. In some the congestion was so marked as to mask the structure. Necrosis or degeneration of splenic reticulum was not observed. There seemed to be no relationship between the degree of congestion and the duration of the experiment.

Kidney: The main finding in the kidney was cloudy swelling of the parenchyma of the collecting tubules. This was more marked in the tissues of the second generation born on the ration. An albuminous precipitate was present in the tubules of some. Congestion also was found. The kidneys of Lot 26 were most affected. In places the cells of the tubules appeared skeletonized, and the glomerular tufts were slightly shrunken.

The results of the histologic examination are shown in Table 3. Lots 5, 6, 7, 8 and 9 were fed on the ration when half grown. Lots 15, 16, 17, 18 and 19 were born on the ration. Lot 26 was born from Lot 15.

GANGRENE OF TOES IN AN INFANT, DUE TO SCALD*

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BOSTON

The case reported herewith is of interest first, because of the etiology, and, secondly, because of the satisfactory end result of conservative treatment.

REPORT OF CASE

History.—An infant, 5 days old, was seen in consultation for gangrene of the toes of the left foot which had resulted from a scald during delivery. The mother had been advised to sit over a pan of hot water to hasten parturition. While doing so a foot presented and was scalded.

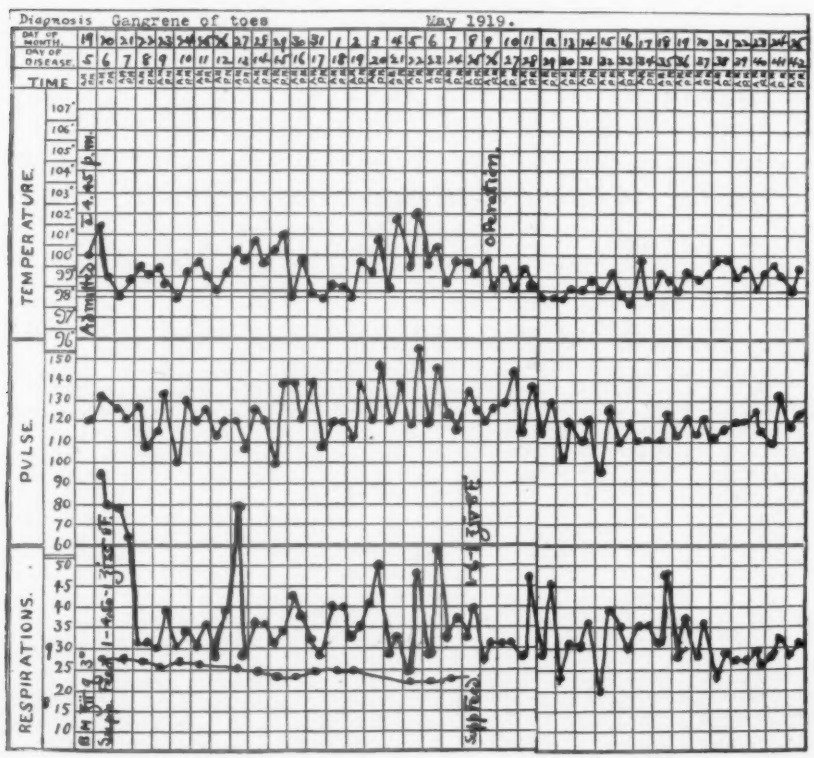

Fig. 1.—Clinical record.

Examination.—The left foot showed a scald of the second and third degree extending up above the ankle on the anterior surface. Gangrene extended to Chopart's line. The infant appeared otherwise normal and was not prostrated.

* Received for publication, Nov. 15, 1920.

* From the Surgical Clinic of the Children's Hospital.

Fig. 2.—Appearance of the foot at entrance to hospital five days after scald.

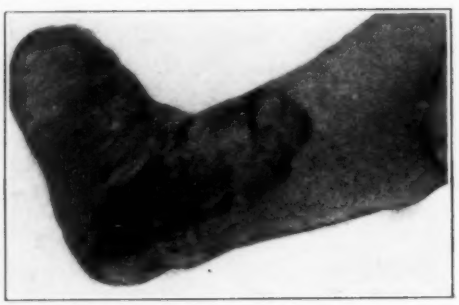

Fig 3.—Cast of toes sloughed twelve days after scald.

Fig. 4.—Dorsal view of slough.

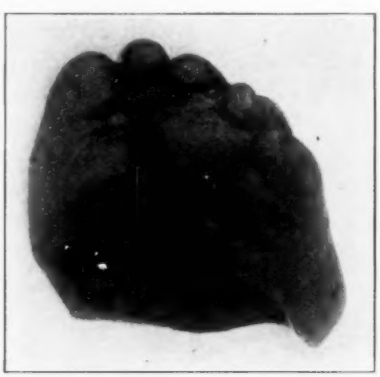

Fig. 5.—Plantar view of slough.

Fig. 6.—Proximal end view of slough showing ends of phalanges.

Fig. 7.—Print from a roentgenogram to show separation of phalanges from foot.

Treatment.—It was considered advisable to send the baby to the Children's Hospital for treatment. After consultation with other members of the staff it was decided to proceed conservatively, avoiding amputation if possible. The foot was placed on sterile towels for open air treatment, and attention was directed to the patient's general condition.

The baby entered the ward May 19, 1918, and its condition was satisfactory after the second hospital day. One week later, the five toes came off as a cast, leaving a healthy granulating surface. Feedings were well taken and normal weight was maintained. The first week in June, a swelling was noticed above the right knee, which increased in size fairly rapidly and became fluctuant. It was assumed to be a metastatic abscess. The roentgen-ray examination of the knee and thigh showed no bone involvement. The abscess was incised and drained under ether anesthesia June 9. A large amount of pus was evacuated. By June 16 there was no more drainage. June 25 the patient was discharged with the incision in the thigh healed and the granulating surface on the left foot was healing well under boric acid ointment dressings.

Result.—A note from the mother, dated August 6, states that the foot is entirely healed. The baby weighs 11 pounds and is well and happy.

COMMENT

Infectious and noninfectious gangrene may occur in infants and children. It may be caused by (1) traumatism of various kinds, including the accidental strangulation of parts; (2) local action of chemicals; (3) disorder of the nervous system, as in Raynaud's disease; (4) constitutional diseases, i. e., hemophilia, purpura and diabetes; (5) thrombosis and embolism; (6) infection by bacillus of malignant edema or other organisms; (7) a combination of morbid influences, often a mixed infection, complicating or following such diseases as scarlet fever, measles, diphtheria, varicella, erysipelas, typhoid, and other fevers; (8) ecthyma gangrenosa. In short, as Kelley states,[1] the possible causes in children include all those which produce the disease in adults, except senile changes.

The interesting point in the case reported is the conservation of a useful foot by allowing the gangrenous tissues to slough rather than removing them by amputation. The abscess of the right thigh was presumably metastatic from the slightly infected granulations of the stump, as the umbilicus was clean and no other focus of infection was present.[2]

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1. Kelley, S.: Surgical Diseases of Children, 1914, p. 148.
2. The following references may also be consulted:

Michael, M.: Am. J. Dis. Child. **20**:124 (Aug.) 1920.

Graham: Diseases of Children, Philadelphia, Lea & Febiger, 1916, pp. 601, 580, 125, 649.

Kerley: Practice of Pediatrics, Philadelphia, W. B. Saunders Co., 1914, pp. 356, 496.

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Stelwagon: Diseases of the Skin, Philadelphia, W. B. Saunders Co., 1915, p. 427.